ARCHIVES OF OPHTHALMOLOGY

VOLUME 22 (old series Volume 79) DECEMBER 1939 NUMBER 6

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CLINICAL DETECTION OF EARLY CHANGES IN THE VISUAL FIELD

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As a rule it is only when defects of the visual field develop slowly that their initial stages can be examined. The study of the methods for the detection of early changes is therefore confined to conditions in which the onset is gradual, such as pressure from a tumor, chronic toxic amblyopia and chronic glaucoma, and is best undertaken with eyes in which the presence of defects is suspected although visual symptoms have not been noticed by the patient. The apparently normal field in cases of supposedly unilateral glaucoma or toxic amblyopia and the fields in cases of enlargement of the pituitary body in which there are no visual symptoms afford suitable material for investigation.

These incipient changes can be elicited only by weak stimuli, such as are capable of disclosing a slight lowering of visual function in the affected area. In this connection two questions arise: First, what are the best and most practical methods of obtaining suitable stimuli? Second, in what part of the field are the earliest signs of functional impairment most likely to be capable of demonstration?

A weak stimulus may be obtained in various ways. Of these the most generally useful is that of reducing the visual angle subtended by the test object by increasing its distance from the eye and diminishing its diameter. Other methods, such as altering the color of the test object or lowering the illumination under which the examination is conducted, are valuable under certain circumstances and may be used in conjunction with reduction of the visual angle.

Figure 1, which represents values obtained by white test objects against a black background, shows that the edge of the field slopes steeply on the temporal side and is perpendicular on the nasal side. It is therefore unlikely that functional impairment of the field will be detected by test objects which are large enough to show the outer limits of the field, since a considerable reduction in the size of the test object will cause only a slight restriction of the field on the temporal side and no change on the nasal side. Such a slight restriction might well be

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 5, 1939.

within the limits of error of perimetric examination. Examination must therefore be conducted at a higher level in the field at a point where a lowering of function will result in a more definite and pronounced alteration in the limits of the field. This condition will be satisfied if the field is examined where the slope of its surface is more gentle than at the periphery, that is to say, about the level which is obtained by a 3 mm. test object (3/330 = 0.5 degrees). A 1 mm. object, which gives an isopter lying in the most gradually sloping part of the field, should then be used. When time is important the 3 mm. test may be omitted. The 1/330 test is reliable, and it must be extremely uncommon for the isopter which it gives to remain unaltered while a more peripheral isopter is affected.

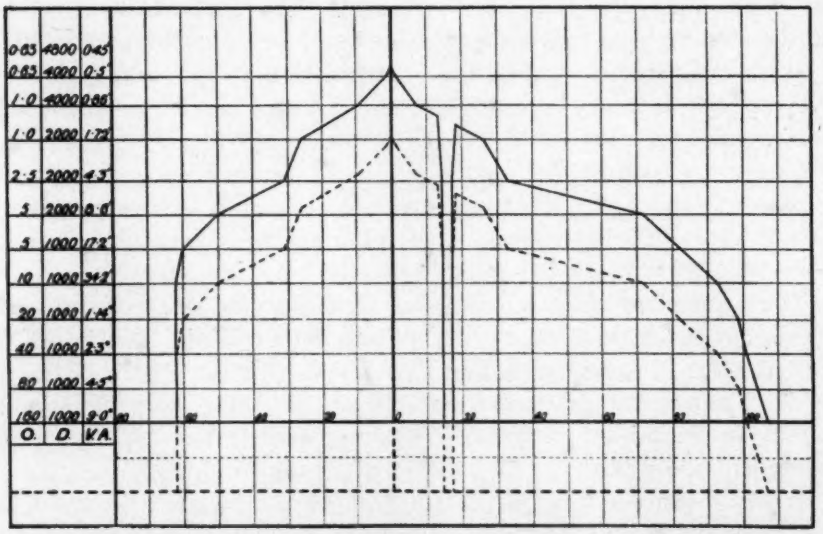

Fig. 1.—The field of vision regarded from the point of view of visual acuity. The slope of the field is much steeper on the nasal than on the temporal side and the most sloping parts are within 30 degrees from the center and more peripherally, between the 35 and 70 degree circles, corresponding approximately to the isopters for 1/2,000 and 1/330. In the case of depression of the field the greatest difference in the width of the isopters is present at the most sloping parts. At the extreme periphery, for example, dividing the size of the test object by 4 reduces the field only by about 8 degrees on the temporal side and not at all on the nasal side, whereas if the diameter of the test object at the level of the 1/330 isopter is halved the field is reduced by about 35 degrees. In this figure and in figure 2, *O.* indicates object; *D.* distance, and *V. A.*, visual acuity. (Published with permission of Henry Kimpton, London.)

In examining the field of vision a clear idea must be present in the mind of the observer as to the nature of the effects of functional impairment. If the periphery of the field is found to be restricted, the field is said to be contracted as if, as expressed in some textbooks of ophthalmology, there were a "pushing in" of the boundary at that

part. Thus the expression "contraction of the field" has come to occupy a definite place in perimetric findings, as if it expressed a peripheral loss of function representing a corresponding impairment of peripheral nerve elements. Such a conception omits the consideration of the condition of the remainder of the field within the periphery. It is as if the coastline of an island were being invaded by the sea, the surface remaining at the same level.

If, however, a more central part of the field is examined where the slope is also gradual, it will usually be found in a pathologic condition that restriction of isopters exists here also. Sometimes, indeed, early changes are more easily elicited in this area. For this purpose the 1/2,000 test is useful. The isopter which it gives extends normally to about 26 degrees from the center in all meridians and surrounds

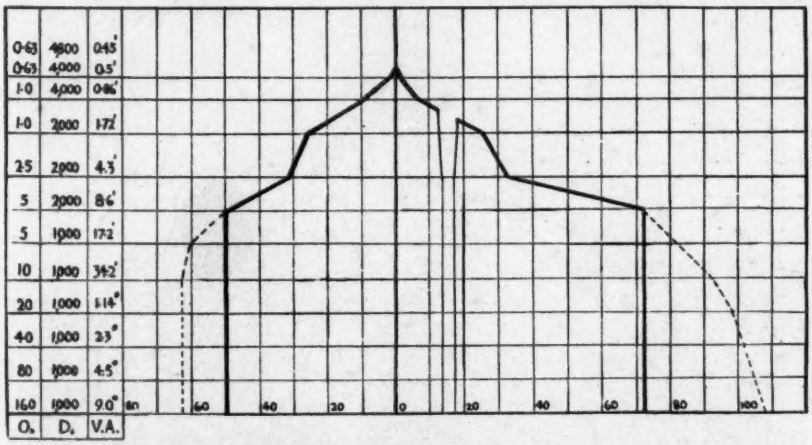

Fig. 2.—Scheme showing the effect of pure contraction of the visual field. The sides of the field are perpendicular up to the point where contraction ceases. Within this point visual acuteness is normal, and the isopters retain their positions. Beyond this point increase in the size of the test object makes no difference. This form of field alteration occurs only in such conditions as hemianopia.

the blindspot about 1 degree beyond the area of absolute blindness. In practice this isopter is one of the most useful in perimetric work, and in cases of early involvement it should always be examined. The 1/2,000 test is simple, reliable and easily applied and deserves a wider appreciation than it receives at the present time. I have found in consulting practice that in the majority of cases in which there has been difficulty in arriving at a conclusion the 1/2,000 test has not been used and that the problem is frequently greatly simplified and sometimes indeed immediately solved by its use.

The presence of restriction of the isopters in the field which lies between the center and the periphery indicates depression of the entire

field. In this case one may imagine an island sinking into the sea so that its circumference becomes restricted, its level being at the same time depressed. The contraction is by no means a "pushing in" of the outer boundary; it is merely the manifestation of the depression. There is therefore a definite distinction between contraction of the field and depression of the field. This distinction is not a mere pedantic quibble. It is a distinction which is of value to the perimetrist in enabling him to develop the correct attitude toward changes in the visual

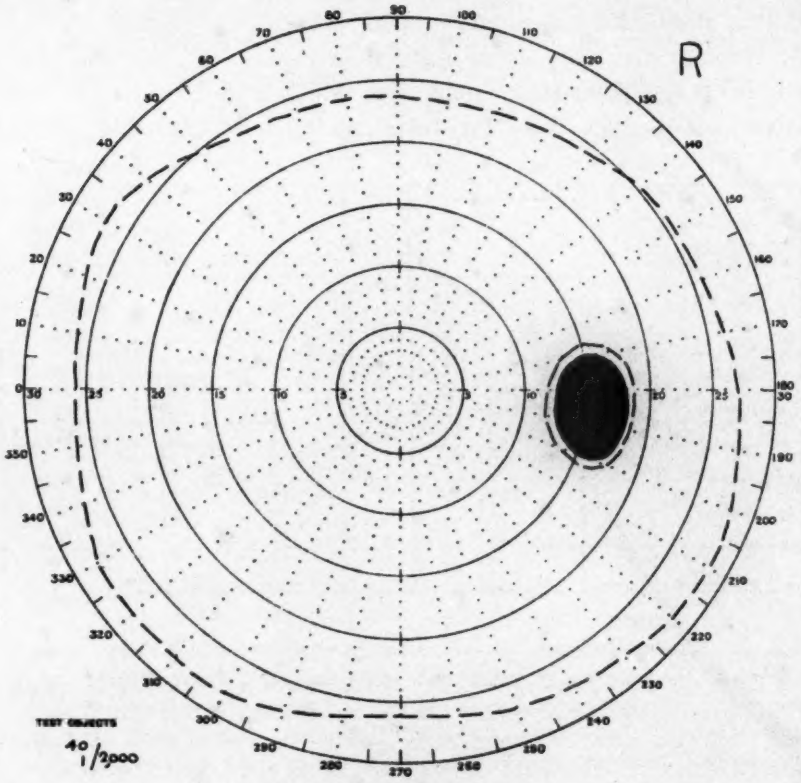

Fig. 3.—The 1/2,000 isopter.

field. Appreciation of this distinction facilitates the correlation of changes in the visual field with the underlying pathologic factors and, in regard to early changes, helps to correct the tendency to focus attention on the periphery of the field. If contraction alone is present, which, apart from conditions such as homonymous hemianopia, is rare, the field within the abnormal boundary is unaltered. It is not so much the size of the field as its condition which one wishes to know. In depression the internal as well as the peripheral isopters are displaced, and in searching for the early signs of perimetric alteration it is there-

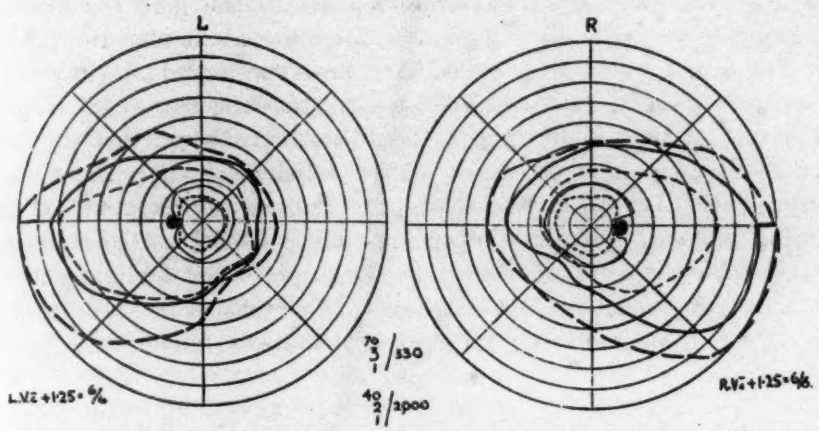

Fig. 4.—Visual field in case of tabes and commencing atrophy of the optic nerve, showing contraction and depression of the fields.

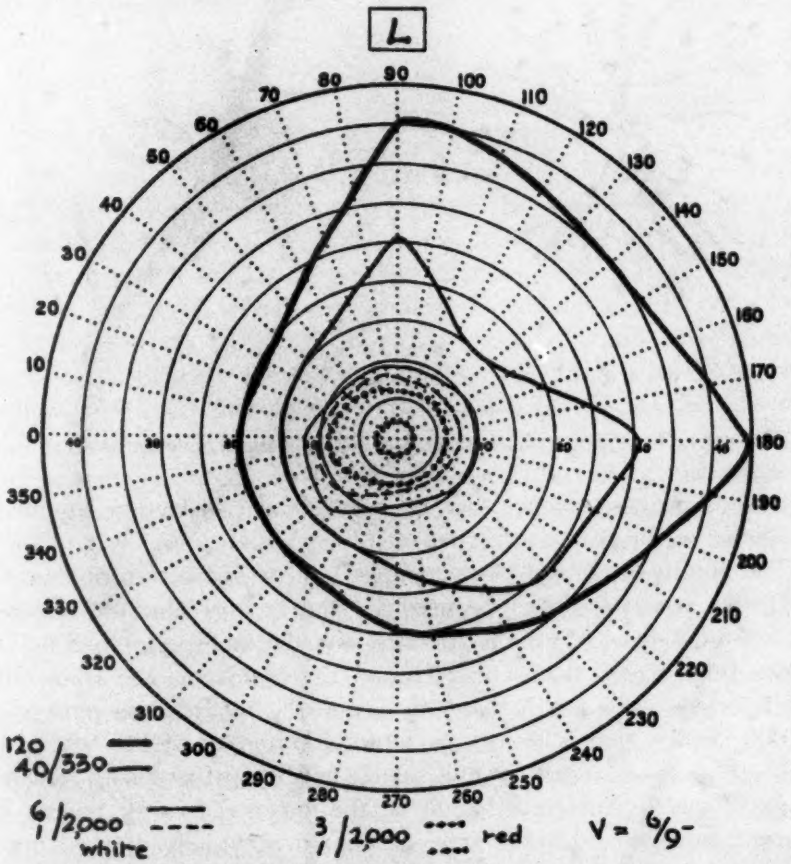

Fig. 5.—Visual field following operation for glaucoma, showing contraction and depression. The field for 1/2,000 white is contracted, but the fields for 3/2,000 and 1/2,000 red are not affected.

fore essential to examine the whole surface of the field for evidence of slight depression. It is most advantageous to examine the field in the first instance at a level where its slope is most gradual, since slight depression at such a part will produce a greater deflection of the isopters concerned. At the same time it should be remembered that the same factor—the gradual slope, which makes pathologic changes more easily elicited—also increases the liability to errors of observation of one kind or another, so that too much importance should not be attributed to slight alterations in the extent of the isopters, but attention should be mainly directed toward alterations in their shape. A reduction in the 1/2,000 isopter to about 15 degrees, if more or less uniform, might

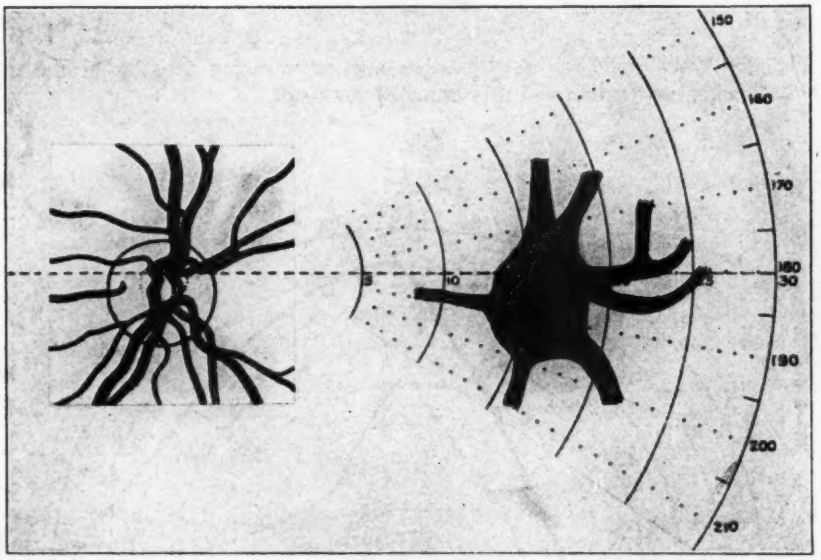

Fig. 6.—The blindspot with vessels. The 40/2,000 and 1/2,000 tests were used.

well be of no consequence, but an even smaller reduction confined to one sector would arrest attention.

The blindspot is most easily mapped out on the screen at 2 meters. A 40 mm. test object may be used for the area of absolute blindness, and one of 1 mm. is usually the best for the amblyopic zone. If the isopter for 1/2,000 does not surround the blindspot, a 2 mm. object may be used. The amblyopic zone should be regarded as part of the 1/2,000 isopter, that is to say, an internal boundary of the field at this level. It is easy to mark out and should not be confused with the vessel scotomas, which are found mainly at the upper and lower poles. The blindspot and its amblyopic zone can be so accurately marked out on the screen that observations made by different persons or at different times will usually be found to coincide to within a small fraction of a degree.

Should it be wished to reduce the visual angle still farther in order to scrutinize carefully the central part of the field, it is more satisfactory to increase the distance to 3 or 4 meters than to reduce the diameter of the object below 1 mm. In this way faint or minute scotomas within about 10 degrees from the center may be examined.

Reduction of the strength of the stimulus by altering its color is usually of more value in estimating the nature of changes in the visual field and in forecasting their progress than in detecting their earliest appearance. One can recollect few cases in which changes were discovered by tests with colored objects which could not be found by tests with white objects with the screen. The value of color testing depends on its power to indicate the nature rather than the presence of altered function, as in the demonstration of the partial and diffuse character of the impairment in an affected area. Thus a colored test object is suitable for the detection of a condition such as tobacco amblyopia.

It is necessary to guard against the assumption that color stimuli have a specific diagnostic value referable to the hue itself. Defects for red and green are not specially indicative of a toxic cause. Little if anything is to be gained by routine color testing, which indeed has been characterized by some observers as a waste of time. On the other hand, in cases of edema of the retina blue may be more poorly seen than red; it is sometimes called green while red is well recognized. This distinction is of value in cases of choked disk with impairment of vision in which it may be possible to show that the impairment is due to the extension of the edema into the macular area rather than to a lesion elsewhere in the visual path. A similar response is obtainable in cases of traumatic edema of the retina, at the shallow edge of a detachment near the macula and in some cases of choroiditis.

Color testing is mainly useful for the demonstration of proportion and disproportion in connection with the fields for white and color. It should not be regarded as a different kind of perimetry, for example, as "qualitative" as opposed to "quantitative" perimetry, but as a part of "quantitative" perimetry, the color factor being regarded mainly from the point of view of reduction in the intensity of the stimulus. It differs from perimetry with white test objects in so far as the test with a small white object stimulates a minute area of the retina with a relatively bright stimulus, whereas the color test stimulates a larger area of the retina with a less intense stimulus.

In the search for early changes in the visual field, even with graded test objects and reduced illumination, I have rarely been able to obtain evidence from color testing which was not already provided, or at least suggested, by small visual angles with a white test object. In some

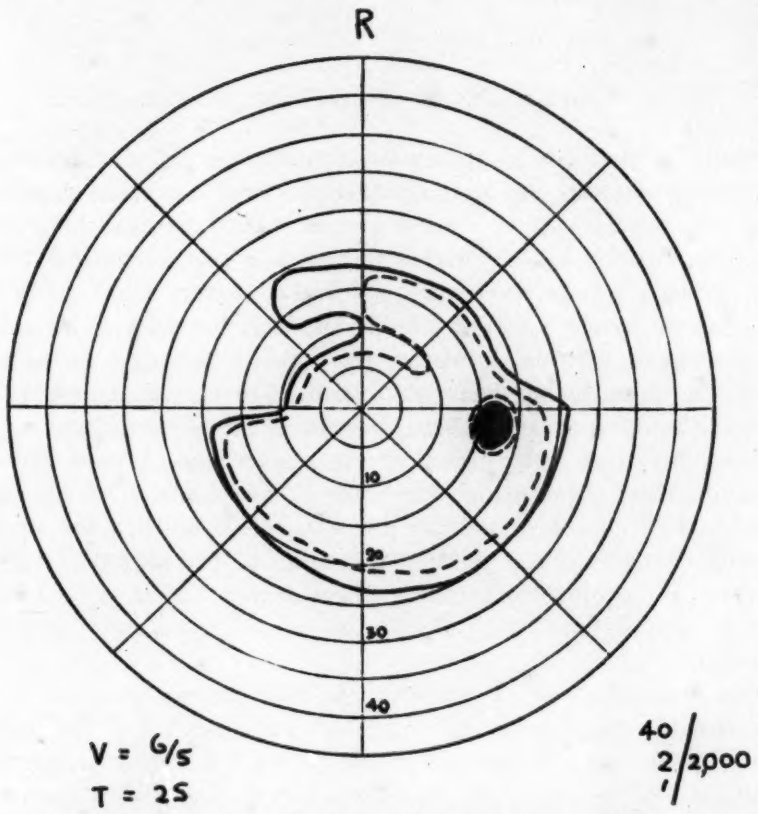

Fig. 7.—Visual field in a case of choroiditis. The defect for blue is as large as the defect for red. The blindspot is enlarged owing to papillitis.

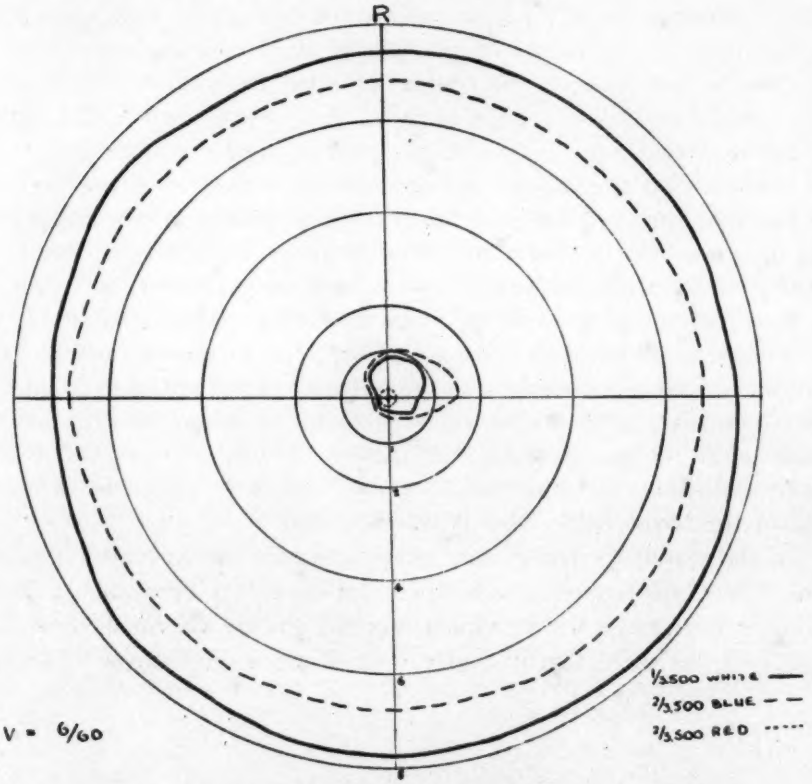

Fig. 8.—Visual field in a case of eclipse scotoma of the right eye.

cases of early involvement color testing may be a useful complementary or confirmatory method, as, for example, in cases of early chiasmal lesions.

Reduction of illumination is sometimes useful, though here again it is perhaps of more value in the amplification of defects, the presence of which is already known or suspected, than in the discovery of incipient changes. It is necessary to reduce the illumination until the fixation object is only just visible to the observer's eye. Slight or even considerable reduction in illumination by drawing the blinds until the room is only dimly lighted is, as a rule, of little avail in the attempt to elicit early changes which elude detection in other ways. The effect of reduction of illumination is more pronounced when the screen is used, especially at greater distances, as, for example, with a 1/3,000 or 2/3,000 test.

The advantage of the screen test at 2 meters depends on the small visual angles obtainable and the enlarged projection of defects which makes them easy to detect, to outline accurately and to analyze. Want of precision owing to unsteady fixation is diminished. It is an easy procedure to carry out and takes no more time than other methods. It must be emphasized that an examination at 1 meter with test objects 2 to 5 mm. in diameter can disclose only defects which have passed the initial stage and will fail to elicit incipient changes. Short range instruments, even with objects as small as 0.25 mm. in diameter, give visual angles considerably larger than can be obtained at 2 meters. A method which does not include the use of a visual angle of 2 minutes or less (1/2,000 = 1.72 minutes) cannot be regarded as adequate any more than a test card would be regarded as adequate which had no lines below 6/12.

Slight variations in the extent of the isopters for small visual angles may be neglected, but variations in shape are significant. This consideration is so important that one feels that no apology is required for its repeated emphasis. The question of size is especially applicable to the blindspot. It has for long been maintained that enlargement of the blindspot is an early sign in connection with lesions of the visual nerve path, especially with regard to glaucoma and diseases of the optic nerve. This statement is based for the most part on observations made by methods incapable of demonstrating really early changes or made at too late a stage in the development of the defect to detect the initial changes. If suitable methods are employed it will be found that the evidence, on which the views so widely held at the present time in connection with what has been called enlargement of the blindspot are based, requires careful scrutiny. It is not enlargement of the blindspot in the sense of magni-

fication of the physiologic defect which is of importance but the appearance of a paracecal scotoma. The expressions "enlargement" or "extension" of the blindspot connote an outgrowth of functional loss spreading from the blindspot into the surrounding field. Careful examination with small visual angles would be necessary in order to establish that this is actually what has occurred when an alteration in the outline of the blindspot is present, and when investigation of this kind is undertaken extension or outgrowth of the blindspot is difficult or impossible to demonstrate. In many cases the extension is really due to the formation of a paracecal scotoma, commonly near the nasal side of the blindspot, which has subsequently become merged with it. In this respect the examination of the amblyopic zone is of importance. The presence of a dense defect adjoining the blindspot cannot be regarded as evidence of an outgrowth from it unless it can be shown that it began at the edge of or inside the amblyopic zone. In such cases it is necessary to search carefully for nuclei or intense areas in the scotoma in which a test object, normally visible in the periphery of the blindspot, cannot be seen, that is to say, an object larger than 2/2,000. If a defect in which such an object cannot be seen can be traced up to the area of absolute blindness in the blindspot, evidence is present that the scotoma is continuous with the blindspot but not that it began by an extension from the blindspot. It is not easy to collect a large number of examples of early changes in the visual field, but such cases in which I have been able to make an examination have indicated the conclusion that most, if not all, scotomas due to conditions such as retrobulbar neuritis, toxic amblyopia or glaucoma, which may appear to have begun by extension from the blindspot, have really originated apart from the blindspot and have subsequently merged with it. Scotomas which arise as extensions of the blindspot are found in conditions such as papilledema, high myopia and juxtapapillary choroiditis. In both groups, as in the case of the periphery of the field, it is alteration in shape rather than in size that is of importance.

The blindspot may also be enlarged in hysteria, in which case the edge of the blindspot appears to behave as if it were part of the periphery of the field.

EXAMPLES OF EARLY STAGES OF SLOWLY ADVANCING DEFECTS

I shall consider some examples of the early stages of slowly advancing defects. The view that the curved scotoma of glaucoma commences as an outgrowth from the blindspot was based on statements by Fleischer and Seidel, who used visual angles of from 10 to 24 minutes. With such large visual angles the amblyopic zone of the blindspot would not be elicited nor could the early stages of

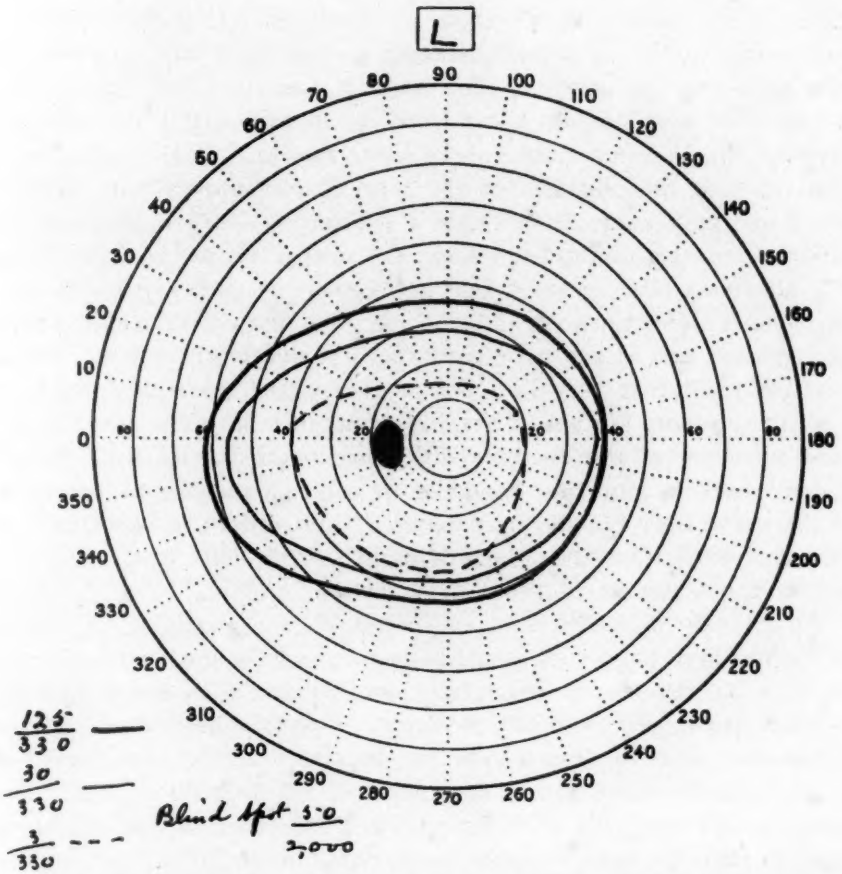

Fig. 9.—Visual field in a case of hysteria, showing enlargement of the blindspot and depression of the field. Glaucoma was suspected. The optic disk showed a wide and deep physiologic cup. Tension was normal.

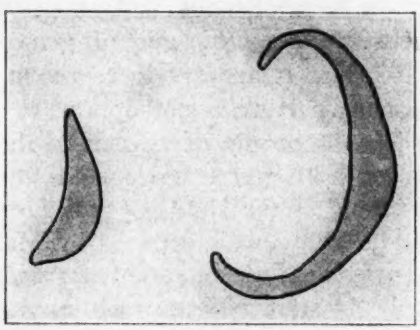

Fig. 10.—Seidel's scotoma. Ths scotoma is continuous with the dense part of the blindspot. It tapers distally, and its curvature is not in accordance with the course of the nerve fibers.

a juxtacecal or paracecal scotoma be detected. Yet Fleischer and Elliot, who used a visual angle as small as 3.5 minutes, believed that if in a case of suspected glaucoma the blindspot were not enlarged glaucoma was not present. Although it has since been shown that the scotoma may develop apart from the blindspot, the old view survives. With a visual angle of 1.72 minutes (1/2,000) I have never been able to find changes of the type of Seidel's scotoma in early glaucoma, and, apart from Seidel's publication, such defects are not reported in the literature. Since the visual angle used by Seidel was about six times greater than is necessary to demonstrate incipient changes, it is legitimate to assume that the scotoma of Seidel should be regarded not as an early but as a well developed defect. It may also be noted that Seidel's scotoma is depicted as being continuous with the absolute portion of the blindspot, that no pericecal amblyopic zone is shown and that the curved defect is pointed at its distal end and wider where it joins the blindspot in contradistinction to the defect of the nerve fiber bundle, from which it also differs in curvature. It seems probable that the Seidel scotoma corresponds in part, at any rate, to the denser portion of a larger arcuate scotoma.

In the case of commencing or suspected glaucoma in which examination has been done with visual angles sufficiently small to show early changes no reliable evidence has been produced to show that the scotoma grows out from the blindspot. Careful observation indicates rather that it arises apart from the blindspot and grows toward it. What is commonly found is depression of the 1/2,000 isopter. This depression is especially noticeable toward the outer side of the blindspot, so that the isopter passes to its nasal instead of to its temporal side. The blindspot is, as it were, laid bare on its temporal side, as a rule on its upper and outer part. This baring of the blindspot is frequent in the apparently unaffected eyes in cases in which the other eye is definitely glaucomatous and is practically constant in cases in which there are definite signs of glaucoma but no other change in the visual field. It appears to represent the initial change in the field in glaucoma. It does not appear to be pathognomonic of glaucoma, as it depends on the depression of the field of vision in the neighborhood of the 30 degree circle, and this may occur from other causes. The baring of the blindspot was present in field charts recorded by Sinclair in 1906 and is depicted by Malbran in his excellent book on perimetry, but little attention has been paid to it, as it can be detected only by test objects such as 1/2,000 or 2/2,000, which have been little used. It may be associated with a small nasal step, which is usually, but not always, found on the side on which the blindspot is exposed, generally on the upper side, as the blindspot

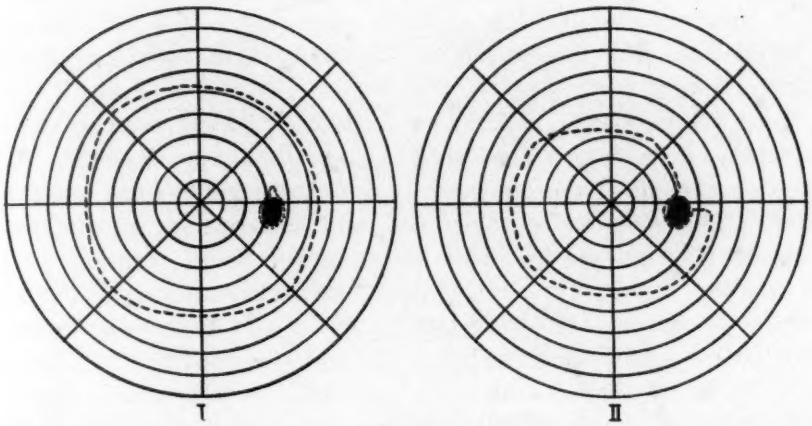

Fig. 11.—Visual fields in a case of early glaucoma. When *I* was taken, halos were present. The tension was 27 and vision 6/6 +. The isopter for 1/2,000 is contracted, and the blindspot is bared upward and outward. After treatment with physostigmine salicylate was begun the halos disappeared. When *II* was taken, tension was 18 and vision 6/6 +. The isopter for 1/2,000 occupies its normal position. The amblyopic zone of the blindspot is intact.

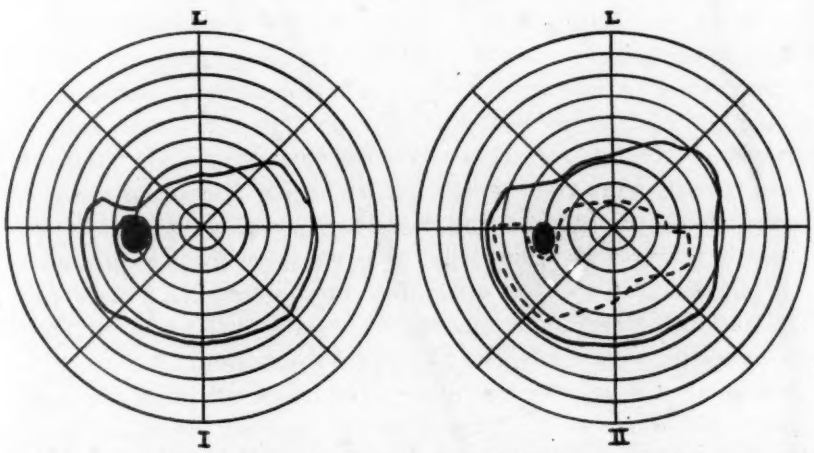

Fig. 12.—Central field of the left eye of a patient who had well developed glaucoma in the right eye. When *I* was taken, tension was 35 and vision 6/6, and the optic disk was normal. The field for 2/2,000 shows depression, especially above, almost merging into the amblyopic area of the blindspot. The field for 1/2,000 is much restricted. In *II* is shown the same field two years later after treatment with miotics. Tension was 21 and vision 6/6. The disk was normal. The field for 2/2,000 has extended, and the field for 1/2,000 shows baring of the blindspot and a small nasal step. The peripheral field was normal on both occasions.

is more commonly bared toward the upper pole. It would be rash to claim that these features indicate more than a local weak area in the field, but it is possible that they may indicate the part of the field in which an arcuate defect of the nerve fiber bundle will appear later. In cases in which glaucoma is suspected I feel that there can be little doubt that this baring of the blindspot has some significance as an early sign.

Tobacco amblyopia is another condition which presents slowly developing defects in which early changes may be studied. Here, also, enlargement of the blindspot is commonly stated to be the initial factor. The scotoma is sometimes said to arise as an outgrowth from the blindspot in the shape of an index finger pointing toward the fixation area. This is because the horizontal meridian passes through the upper part of the blindspot, and if a narrow scotomatous area exists on the horizontal meridian and is joined to the blindspot the index-finger shape results. If, however, the scotoma is carefully examined it will be found that this narrow finger has a central dense core or that toward its outer end there is a small dense nucleus, indicating that the defect commenced as a small scotoma situated on or about the horizontal meridian to the inner side of the blindspot, subsequently enlarging and merging with it. I have not been able to find any recorded case which clearly showed that the defect was truly an outgrowth from the blindspot.

In cases of tabetic atrophy changes in the visual field of various kinds develop slowly. Central vision may be normal, and the optic disk may show a healthy color, though commencing changes in the field are present. Two well known types are concentric and sector contractions. In both of these types it will be found that the central isopters participate in the change, and frequently the use of the 1/2,000 test constitutes the most simple and easy means of ascertaining that the field is not normal. Similarly, after recovery from conditions such as retrobulbar neuritis, the optic disk may appear pale with normal central vision. Here again this test will show that pallor of the disk is not compatible with integrity of the field of vision.

Slowly advancing changes in the visual field are also produced by pressure from tumor. This may affect any part of the visual pathway. In the case of the optic nerve, a tumor may grow from its sheath or may arise in the orbit apart from the nerve. The tumor produces exophthalmos, and, especially in the cases in which ophthalmoscopic appearances are normal, it is of interest to know whether the optic nerve is involved or not. Here again it will be found that although the pressure affects the nerve presumably first at the periphery it is the more central regions of the field which are first affected. This is well brought out by Malbran, whose charts of the field in cases of

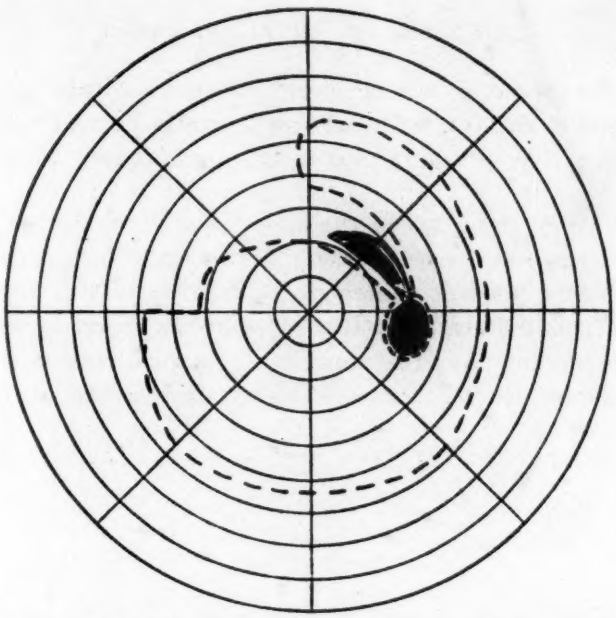

Fig. 13.—Visual field of a patient with tension of 15 and vision of 6/6 +. The optic disk was deeply cupped. Note the arcuate scotoma with a nasal step for the 1/2,000 test. The denser part of the arcuate scotoma is not continuous with the blindspot. Had the 1/2,000 test not been used, the full extent of the arcuate scotoma and the nasal step would not have been shown.

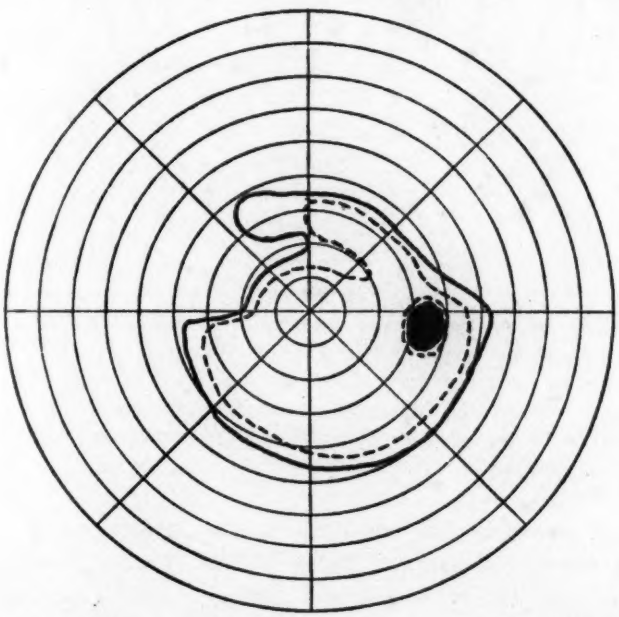

Fig. 14.—Visual field of a patient with early glaucoma. There are depression of the central field and a small nasal step with a partial arcuate scotoma which is advancing toward the blindspot but is not in contact with it. The amblyopic zone of the blindspot is intact. The tension at the time the field was taken was 25 and the vision 6/5.

orbital tumor show a normal periphery with contraction for tests from 1/2,000 to 5/2,000 with normal or nearly normal central vision. In other cases a central scotoma may be present with a normal periphery.

In the case of the trunk of the optic nerve there are few perimetric indications as to the site of a lesion. It is usually believed that lesions of the periphery produce concentric contraction of the field of vision while lesions in the center of the nerve produce central scotoma. It has been seen, however, that so-called concentric contraction usually affects the inner parts of the field of vision also, indicating that the

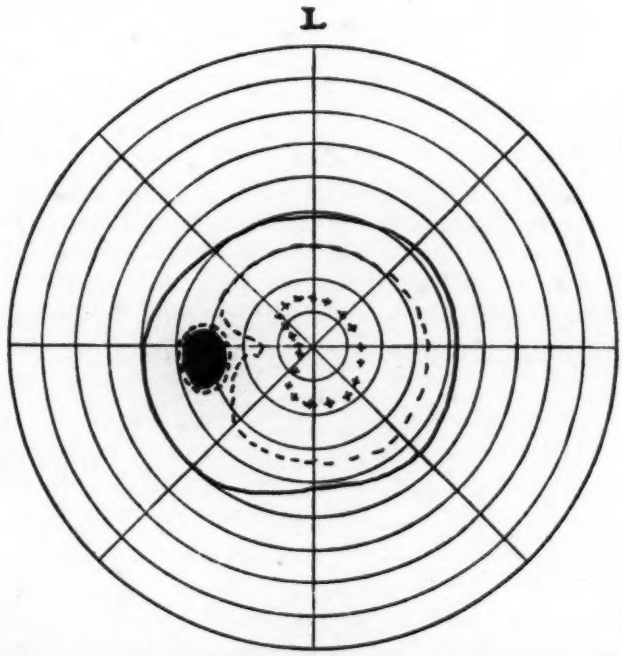

Fig. 15.—The central field of the apparently normal left eye of a patient with definite early tobacco amblyopia in the right eye. The isopter for 2/2,000 (unbroken line) is contracted and that for 1/2,000 (broken line) is even more contracted. The amblyopic zone of the blindspot is intact. The region of the centrocecal scotoma is shown by an indentation in the 1/2,000 isopter and also in the isopter for 10/2,000 red (crosses).

lesion in the nerve is not confined to the periphery. At the distal end of the nerve the papillomacular bundle is superficial, and it has been pointed out by Wilbrand and Saenger that a peripheral lesion here may produce peripheral contraction of the field together with centrocecal scotoma. As regards the portion of the nerve from the point where the papillomacular bundle becomes central to its junction with the chiasma, there are no perimetric signs which indicate how

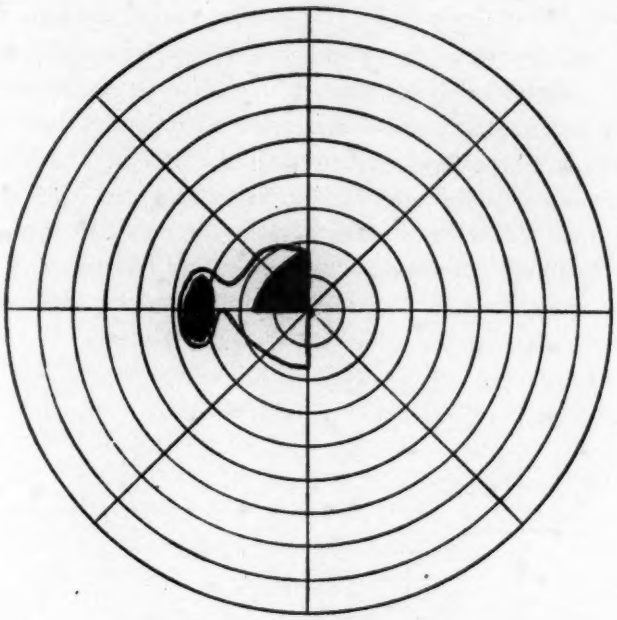

Fig. 16.—Junction scotoma.

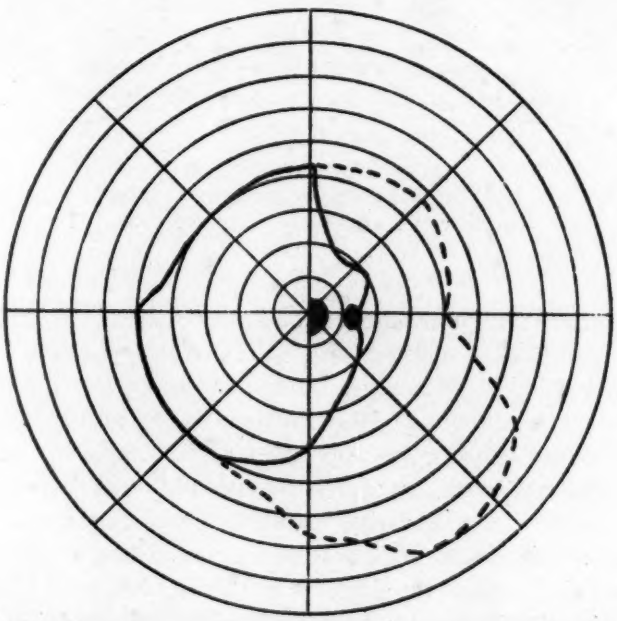

Fig. 17.—Unilateral hemianopic central scotoma (junction scotoma) with depression of the temporal field. The field of the other eye was normal. Two years later temporal hemianopia developed also in the other field.

far along the nerve a lesion may be situated. At the chiasmal termination of the nerve the crossed and uncrossed fibers separate, and a small lesion may occur at this point and affect the crossed fibers only, producing a unilateral temporal hemianopic or quadrantic central scotoma called "junction" scotoma, since it indicates the site of the lesion at the junction of the optic nerve and chiasma.

The junction scotoma may develop rapidly as the result of disseminated sclerosis. Sometimes it is caused by pressure from a tumor and appears gradually, leading in the long run to bitemporal hemianopia. In one case the diagnosis of pressure on the inner side of the right optic nerve from a tumor was not accepted at first, and alternative diagnoses, including hysteria, were advanced, but two years later the presence of bitemporal hemianopia was definite. In such cases the

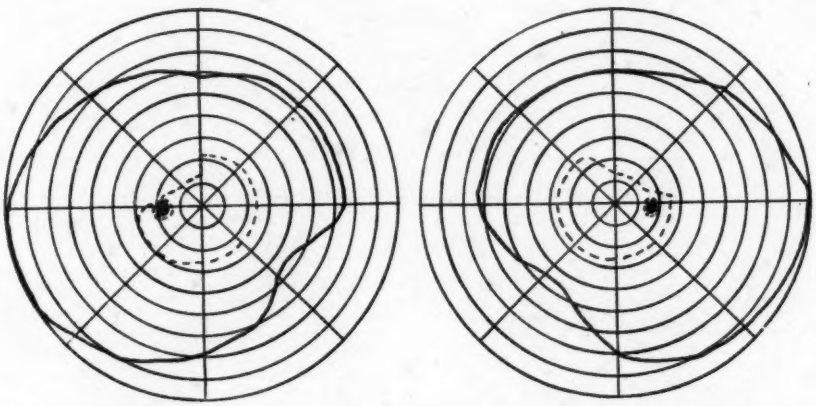

Fig. 18.—Visual fields in a case of commencing chronic acromegaly. The central fields show definite upper and outer quadrant disks. The periphery was normal for objects as small as 1/330.

correct diagnosis can be reached if both fields are examined at intervals by the quantitative method with several test objects.

As regards localization, one is on firmer ground in connection with the chiasma. The changes of bilateral hemianopia are well known and are usually described as commencing in typical cases by the appearance of a temporal slant or cut in the periphery of the upper outer quadrant. In slowly progressive acromegaly the fields are said often to be normal. If the 26 degree isopter is examined in such cases in which vision is normal but chiasmal interference nevertheless may be present, it will frequently be found to be deflected inward in its upper outer quadrant before the periphery of the field shows alteration. This feature has also been observed by Malbran, and it cannot escape observation by any one who uses the 1/2,000 test. One would

be reluctant to accept the statement that the fields were normal in a case of acromegaly unless it had been ascertained that the isopters within the 30 degree circle had been shown to be unaltered. It is this feature in the development of bitemporal hemianopia which renders unacceptable the view that the restriction of the temporal field sometimes found in pregnancy is due to the physiologic enlargement of the pituitary body. In pregnancy the pituitary body enlarges gradually, and if it were to extend upward far enough to press on the lower surface of the chiasma it would produce the same changes in the visual field as are caused by a slowly growing intrasellar tumor; but while peripheral temporal restriction, affecting the lower as well as the upper temporal quadrants, has been reported, characteristic alterations in the central isopters have never been demonstrated in cases of uncomplicated pregnancy. In order to establish the presence of true bitemporal hemianopia, it is necessary to examine the whole field, including the more central isopters.

Since bitemporal hemianopia is rarely symmetric in the earliest stages, it may be expected that one field will be affected while the other remains normal. Where the 26 degree isopter in one field only is deflected in its upper outer quadrant, it would be difficult to say whether a homonymous or a bitemporal hemianopia would ultimately develop.

The same principle applies to the suprachiasmal pathway: Early changes are likely to be found in deflection of the internal rather than of the peripheral isopters. Examples will be found in Cushing's papers on lesions of the temporal lobe. The outline of the 26 degree isopter may, however, be modified by the presence of choked disk, a complication which must be taken into consideration in arriving at a conclusion.

The early failure of the central part of the field, whether in the form of slight depression or of central scotoma, is probably due to several factors, one of which is the blood supply. The blood vessels of the visual nerve path are for the most part so arranged that the central fibers are most remote from nutritive supply, and this is probably one of the reasons that the changes are found near the center of the field. This hypothetic explanation, though not proved, seems more attractive than the view that the more highly developed physiologic function of the central elements is associated with greater vulnerability.

DISCUSSION

Dr. Luther C. Peter, Philadelphia: The name of Traquair and the subject of perimetry are so intimately associated that to know perimetry is to know Traquair. The reverse, however, is not necessarily true. This intimate association is not due to the fact that there are

comparatively few ophthalmologists who are profoundly interested in perimetry but rather to the fact that Mr. Traquair began as a young man to develop the subject and has continued throughout his life to give perimetry major consideration. The society is to be congratulated, therefore, on this opportunity to hear Mr. Traquair discuss phases of his favorite subject in ophthalmology. When I was a young man the dean in one of the medical schools once told me that to try to be an all-round ophthalmologist would only add one more ophthalmologist to the list, but to select even a narrow specialty within the specialty would surely land one at the top rung of the ladder. Mr. Traquair ideally exemplifies this thought.

To add anything to the discussion which Mr. Traquair has so admirably presented would be foolish. It would be an anticlimax. I think our experiences run along the same lines. Probably our methods of approach differ somewhat, but we arrive at the same conclusions. I was brought up on the tangent screen of long radius, but after the introduction of some of the standard perimeters, particularly the Ferree-Rand perimeter, I discovered that in a certain number of instances I could obtain much more satisfactory and accurate information with a radius of 330 mm. than with an instrument of 1 or 2 meter radius.

There are several points on which perhaps we do not altogether agree, and I would not feel that I was doing justice to what I have been teaching in perimetry if I did not stress the importance of color perimetry. There are so few light sense instruments on the market that are of any real value; they are too time consuming. I hope that some time an instrument will be presented that will be practical. Color perimetry, particularly in glaucoma and in atrophy, is a good test for reduction of light sense. The form field may be full, but when colors are reduced I think one will find that the light sense instrument will also show a considerable reduction in the light sense. I should like to call attention to the fact that the pathologic involvement varies in certain conditions. In glaucoma, for example, one is dealing with atrophy of nerve bundles, and in that particular instance I feel that I can detect earlier changes in glaucoma with the good illumination obtainable on an instrument of 330 mm. radius than at a distance of 1 or 2 meters. Color perimetry is valuable in glaucoma, though not quite as valuable as in toxic amblyopia, when green is the most sensitive test that can be used. In atrophy of the optic nerve I believe that form fields alone are adequate to determine the extent of the damage, particularly in the quantitative form to which Mr. Traquair has referred, but I do feel that color perimetry should not be excluded. One is missing too much if one fails to use colors in those instances in which experience teaches that colors as well as form should be used.

MR. H. M. TRAQUAIR, Edinburgh, Scotland: My object in trying to stress the use of the screen at 2 meters is to introduce a simple and practical method which can be used by every one. I think that what is wanted in the practice of ophthalmology at the present time is not so much special methods but a wider adoption by ophthalmologists in general of some simple, practical method, and I think the screen does provide that method.

With regard to color perimetry, I do not exclude it by any means, as will be seen from some of the slides I showed, but I do not think one finds out anything by color perimetry, as I said before, that one has not concluded from the other tests. Color perimetry has certain valuable uses of its own, and it is a helpful complementary method. I think that Dr. Peter and I really agree on this subject. I do not wish by any means to exclude the use of color perimetry from clinical work.

INJECTION OF AIR AS FACTOR IN MAINTAINING FILTRATION AFTER CORNEOSCLERAL TREPHINING IN GLAUCOMA

J. A. MacMILLAN, M.D.
MONTREAL, CANADA

Corneoscleral trephining for chronic glaucoma gives satisfactory results in only about 75 per cent of cases. Any procedure which will increase this percentage is therefore worthy of recording, and I wish to suggest that the routine injection of air beneath the conjunctival flap and into the anterior chamber at operation may be of value in this connection.

My experience leads me to believe that the presence of air beneath the conjunctiva will prevent the adhesion of the flap to the underlying sclera for approximately eight days, as it requires about this length of time for complete absorption of the air. The injection of air into the empty anterior chamber restores it immediately and allows the aqueous to replace the air gradually. This is accomplished in about a week's time.

BENEFITS OF PROCEDURE

I shall first consider the benefits to be obtained from the two effects just stated. As advised by Elliot, the flap for corneoscleral trephining is made by separating the conjunctiva and Tenon's capsule from the sclera, and at the completion of the operation it is replaced with or without sutures. It is only the seepage of aqueous beneath the conjunctiva which creates the permanent bleb. It would seem that by raising the conjunctiva from its bed for a few days while the draining of aqueous from the globe is being stabilized that the adhesions between the flap and the sclera would be prevented over a wider area. In this way a more diffuse bleb will remain, and consequently a discrete and vesicular fibrous scar should be avoided. Elliot[1] thought that the vesicular bleb was probably the result of faulty technic, but I am sure that all ophthalmologists have seen it occur in cases in which it was felt that the technic had been faultless. The water-tight suture advo-

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 6, 1939.

1. Elliot, R. H.: A Treatise on Glaucoma, ed. 2, London, Oxford Medical Publications, 1922, p. 600.

cated by Foster Moore[2] was intended to mitigate the vesicular bleb, and I think that the same can be claimed for the injection of air beneath the flap.

The immediate restoration of the anterior chamber at operation is also to be desired. While the usual time required for the reformation of the anterior chamber after operation is three to four days, many times it is delayed beyond this time, and in rare instances weeks are required before it is accomplished. During this period the posterior synechiae frequently occur, and nothing can prevent them, for with an empty chamber mydriatics have little or no effect. When the chamber is full of air at the completion of the operation the iris is not in contact with the cornea, and each day more aqueous and less air can be seen occupying the chamber. With the presence of the aqueous, mydriatics are absorbed, the pupil can be kept wide and synechiae are avoided. Moreover, in restoring the chamber the iris is separated from the trephine hole and prolapse of the iris during the healing process is prevented.

APPLICATION OF PROCEDURE

While inserting a continuous suture after a corneoscleral trephining in January 1938, I noticed air bubbles beneath the flap. It occurred to me that this might happen frequently and be unobserved and that it might be partially responsible for the advantages claimed for the water-tight suture. It therefore seemed that it would be a good idea to inject air routinely beneath the flap and at the same time to force air into the collapsed chamber. I have since followed this procedure for a number of patients with advanced glaucoma. Injection of air beneath the flap is a simple matter and easily accomplished, but greater difficulty is experienced in establishing a chamber and maintaining it. At first, after completion of the operation and the insertion of the water-tight suture, air was injected beneath the flap with a hypodermic needle and forced through the trephine hole into the anterior chamber by gently stroking the surface of the flap with an iris repositor. This could not always be accomplished. The next procedure was varied by passing a small hypodermic needle from behind the sutured incision up to the trephine hole before injecting the air, but here again not enough air reached the chamber to give it the desired depth. Finally, a fine curved lacrimal syringe needle was passed through the trephine hole before the suture was inserted, and the air was injected directly into the chamber. With the point of the needle directed forward and hugging the cornea while the flap was gently lifted forward, no

2. Moore, R. F.: A Watertight Suture in Trephining, Brit. J. Ophth. **7**:257. 1923.

difficulty was experienced in entering the chamber. In this way a deep chamber was obtained, but care had to be used in suturing the flap to prevent the air from leaking back through the trephine hole. At the completion of the suture more air was injected beneath the flap. I have found that the latter method is the easiest and safest. In one case in which the eye was blind air was injected through a fine hypodermic needle entered at the limbus away from the site of operation. It was not easy to insert the needle between the iris and the cornea when they were in apposition in a soft eye. However, it gave an excellent filling of the chamber, and sufficient air escaped through the trephine hole to raise the conjunctival flap from its bed. This procedure is not advocated except in careful hands on account of the danger of injury to the lens.

Filling of the chamber with air is a natural consequence in softening of the globe. It frequently happens in cataract extractions but seldom after corneoscleral trephining. Fuchs [3] stated that the cornea is pressed in by the pressure of the air—collapsus corneae—and that if it suddenly tends to resume its normal curvature it makes the aqueous chamber fill with air by suction. From this it is evident that the procedure here advocated is only an imitation of what nature suggests. Further, in the preparation of this presentation I found in looking through the literature that subconjunctival injections of air and injections into the anterior chamber have been used for years for other purposes, as in cases of tuberculous iridocyclitis and tuberculous keratitis. Koster,[4] of Leyden, is credited as being the pioneer in this procedure.

The air to be injected should be drawn through a sterile sponge into a sterile glass syringe, preferably one of 5 cc. capacity; but no sterilization of the air itself is needed. The preparation of air in such a way and injection into the cerebrospinal canal is an everyday procedure for the neurosurgeon.

Oxygen was injected into the anterior chamber of rabbits, but it was found to be not as satisfactory as air owing to the rapidity of its absorption. Nitrogen was also used, but it did not seem to have any advantage over air. The number of cases in which air was injected under the conjunctival flap and into the anterior chamber was small, as it was done chiefly in cases of long-standing glaucoma in which there was impairment or absence of vision. But in all, the tension was reduced to normal or lower, the blebs were diffuse and secondary operations have been unnecessary to date.

In two of the first eyes operated on the air beneath the flap seemed to disappear sooner than I thought it should. More air was injected,

3. Fuchs, E.: Consequences of Softening of the Eyeball, Arch. Ophth. **3**:419 (April) 1930.

4. Koster: Klin. Monatsbl. f. Augenh. **40**:340, 1902.

one injection being made on the fourth day and the other on the ninth day after operation. The pressure required to force the air beneath the conjunctiva seemed much greater than when the injection was done at the time of operation. This would indicate that adherence of the flap must take place early. In the other cases a larger quantity of air was injected at the time of operation, so that the conjunctiva was lifted up over the upper half of the globe. Following this there was often a good deal of redness of the conjunctiva, but it was not more than is occasionally found when the procedure is not followed.

The question of injecting air into the anterior chamber postoperatively in cases of delayed reformation of the chamber is a matter that I have had under consideration.

In this article I have considered only the restoration of the chamber and the formation of a diffuse bleb. I have taken for granted that the trephining of the cornea and sclera had been carried out successfully with removal of the whole disk and that none of the iris had remained incarcerated in the opening. It cannot be expected that restoration of the chamber or lifting of the conjunctiva from its bed can overcome the failure of a proper fistulous opening through the corneosclera. If the trephine hole should block and the aqueous fail to leak out, the conjunctival flap will, of course, heal tightly down to its bed after the air has been absorbed.

Many of the observations here recorded have been verified by animal experimentation.

DISCUSSION

DR. JONAS FRIEDENWALD, Baltimore: Dr. MacMillan's ingenious suggestion seems to me to be directed to two of the most difficult aspects of the surgical treatment of glaucoma. Some time ago it was possible for me to show that the opening of the glaucomatous eye by any intraocular operation results in edema of the ciliary body, which is equivalent to an opening of the anterior chamber, and that as a result of this procedure any operation which enters the interior of the eye, especially if the anterior chamber is opened, is accompanied by more or less increase of peripheral anterior synechiae, so that the anatomic basis of the glaucoma gets worse, even though the successful fistulizing operation may lead to permanent control of the pressure. The procedure of injecting air into the anterior chamber to establish a normal intraocular pressure after the operation would seem well directed to avoid this difficulty. It may be of interest to the members of this society that Dr. John McLean, resident at the Wilmer Ophthalmological Institute, has independently arrived at this same procedure, and though I do not wish to anticipate his reports, he has been utilizing this method postoperatively for the past month.

The second aspect of the problem is that of the scarring of the conjunctival flap. In operations which are otherwise technically satisfactory, the chief cause of failure has seemed to me to be increased scar formation about the operative flap. While I have not had any experience in dealing with this condition by injection of air, for the past years

I have made a practice in cases in which recurrent operations are required and in which the previous operations were beyond criticsm from the technical point of view, and in which I therefore believed the cause of the failure of operation to be increased fibrosis of the flap, of using beta irradiation in order to inhibit fibrosis. I have had rather striking success with this form of treatment in a number of cases in which repeated operations for glaucoma had been performed, all of which seemed at the time to be perfectly satisfactory but which failed to control the intraocular pressure permanently.

DR. CONRAD BERENS, New York: Dr. Bogart and I have attempted to do a somewhat similar thing in two different ways. As I have had experience with 150 filtration operations by the method of injecting salt solution underneath the flap, I thought that it might be of interest to find out if Dr. MacMillan had some of the same complications that I have observed. The flap is closed by Foster Moore's water-tight suture, and one-half strength physiologic solution of sodium chloride is introduced under it. No dressing is used. I thought that good results were being obtained. I have tried to introduce my sutures so as to exert traction upward, in an attempt to pull up the flap, because in 3 cases the flap has fallen down over the cornea considerably and has adhered over the sutures. The salt solution does not stay under the flap as long as the air does, if the air stays a week. In a great many cases there has been loss of the anterior chamber. In 2 cases the flap has come down and there has been a tendency for the bleb to extend into the cornea. I should be interested to know if Dr. MacMillan had the same difficulties with his procedure that I have had.

DR. P. C. JAMESON, Brooklyn: I should like to mention an interesting thing in connection with the filling-up of the corneal trephine opening after operation. In doing some work on vascularization of the anterior portion of the eye it was noticeable that to get to the deeper layer of vessels it was essential to remove the capsule, as it was agglutinated to the margins as the cornea was approached. The capsule cannot be curetted at this point. I feel sure that after operation the capsule swells at this site and that this is the reason why the aperture closes. Elschnig has mentioned that he always removes the capsule which is intimately associated with the sclera at the point posterior to where the trephine is made. It is possible to remove it only by cross incisions in this portion. If one takes a cataract knife and makes some cross incisions, one meets with a bulklike tissue which is altogether different from the sclera proper, and only by making these cross incisions with a Graefe knife can it be removed. Elliot made the statement that removal of the capsule is absolutely essential and that the tissues should be removed both from the conjunctiva and from the base of the opening, as far as possible; otherwise the opening will close. I too think it is essential to do that. This procedure always relieves congestion of the ciliary body, because if the capsule is removed at that point by simple incisions and curettage, the vessels of the ciliary body may be seen exuding. The procedure not only relieves congestion but reduces tension. I think it is a good idea to incise the capsule at this point down to the sclera, as in all probability it prevents closing of the aperture.

DR. ARNOLD KNAPP, New York: I should like to ask Dr. MacMillan whether he believes that by following out this technic of injection of air the incidence of choroidal detachment is lessened. I think the prolonged exhaustion of the anterior chamber following a trephining operation is the greatest difficulty and the one which is followed by poorest filtration.

I am particularly gratified and interested in Dr. Friedenwald's observation on the use of beta rays following trephining.

DR. J. A. MACMILLAN, Montreal, Canada: Dr. Berens asked if I noticed any falling down of the flap. I have not. My observations have not been extensive. Such a complication may be possible, but I have not seen it yet.

Dr. Jameson spoke of removing Tenon's capsule. I have never done it, but I should feel that by so doing I should be more likely to thin the flap, and that would not be wise.

Regarding Dr. Knapp's question concerning choroidal detachment, I have not yet looked into any of these eyes immediately after operation. I did not want to disturb the air as I wanted to keep it under the flap as long as possible. I have thought of the question of choroidal detachment, and I believe that restoration of the chamber at the time of operation would probably avoid it.

RETINAL DETACHMENT CURED BY AN EYEBALL-SHORTENING OPERATION

REPORT OF A CASE

D. K. PISCHEL, M.D.
AND
MIRIAM MILLER, M.D.
SAN FRANCISCO

In 1933 Lindner published the description of an operation which he called an eyeball-shortening operation. This procedure is a modification of a similar operation previously described by L. Müller and was suggested by Lindner for use in cases of retinal detachment in which operation by other methods had been unsuccessful. He also suggested and used it for the first operation in certain unfavorable cases. As can be imagined, this operation is most applicable when the detached retina is found to be held away from its normal bed by fine vitreous strands or by agglutination of retinal folds.

The technic of the operation is fully described in Lindner's article [1] and is easily understandable from the illustrations, even if the reader is not proficient in German.

Essentially, the operation consists of excising a long, narrow crescent-shaped piece of sclera lying concentric with the limbus and suturing the cut edges of the sclera together. The length of the excised piece may be from just under half to just over half the circumference of the globe.

Briefly, the technic is as follows:

The sclera is laid bare, and one rectus muscle is cut off. Specially equipped dividers are needed, one leg of which has a dull point and the other a sharp blade. (This instrument can be obtained from Grieshaber, Schaffhausen, Switzerland, or can be adapted from a small drawing dividers by any mechanic.) A line concentric with the limbus and 9 to 10 mm. from it is lightly cut in the sclera by drawing the dividers over it, the blunt end being kept on the limbus and the blade end in the sclera. Next a second line is cut (as is illustrated in fig. 1) posterior to the first one, in a similar manner, the blunt end of the compass being kept on the first cut and a new cut being made with the blade end. These two incisions should be from 2 to 6 mm. apart.

The incisions are deepened with a keratome (fig. 2) through three quarters of the scleral thickness. Care must be taken not to perforate the sclera and choroid, as the eyeball may collapse or severe hemorrhage may occur. Double-armed fine chromic catgut sutures are placed 2 mm. apart through both peripheral lips

From the Division of Ophthalmology, Stanford Medical School.

1. Lindner, K.: Ztschr. f. Augenh. **81**:277, 1933.

of the incisions (in the form of a mattress suture), leaving the flap free. These sutures should be placed at this time, as it is difficult, if not impossible, to do so after the piece of sclera has been entirely excised. After sutures are in place, the crescent of sclera is slowly cut out. First, the two parallel incisions are joined at one end, and then with a fine scissors the remaining layers of scleral fibers at this end are cut through, leaving the choroid bare. Care must be exercised to see that the choroid is free from the sclera. When 2 to 3 mm. have been thus cut through, the first set of sutures is carefully tied (fig. 3), care being taken that the choroid is not caught in the lips of the sclera as the sutures are drawn up. The free

Fig. 1.—The sclera is exposed and the rectus muscle cut. With a pair of special dividers the second line is lightly cut into the sclera, 2.5 mm. behind the first.

end of the flap of sclera is drawn under the other sutures and the flap is cut through farther, the sutures again being tied as more choroid is laid bare. As the process continues and the globe becomes smaller, the choroid will be bulged out through the incision by the pressure from within. The choroid must be punctured with a fine discission needle and some of the intraocular fluid be allowed to escape; the choroid can then be tucked back inside the sclera. In making this puncture one should avoid large choroidal vessels, as otherwise disturbing hemorrhage may occur. After the crescent of sclera has been completely excised and the last scleral suture tied, the cut muscle may be resutured and the conjunctival incision closed.

In areas in which former operations had been performed the choroid will be found adherent to the sclera. Here a thin layer of scleral tissue may be left behind. The operation is tedious and must be done with the aid of a loupe.

Up to the present no case in which eyeball shortening was employed has been reported in the American literature. Lindner [1] in his original article, reported cure in 1 case in which the operation was done and improvement in 3. Ramach [2] reported healing in several cases from

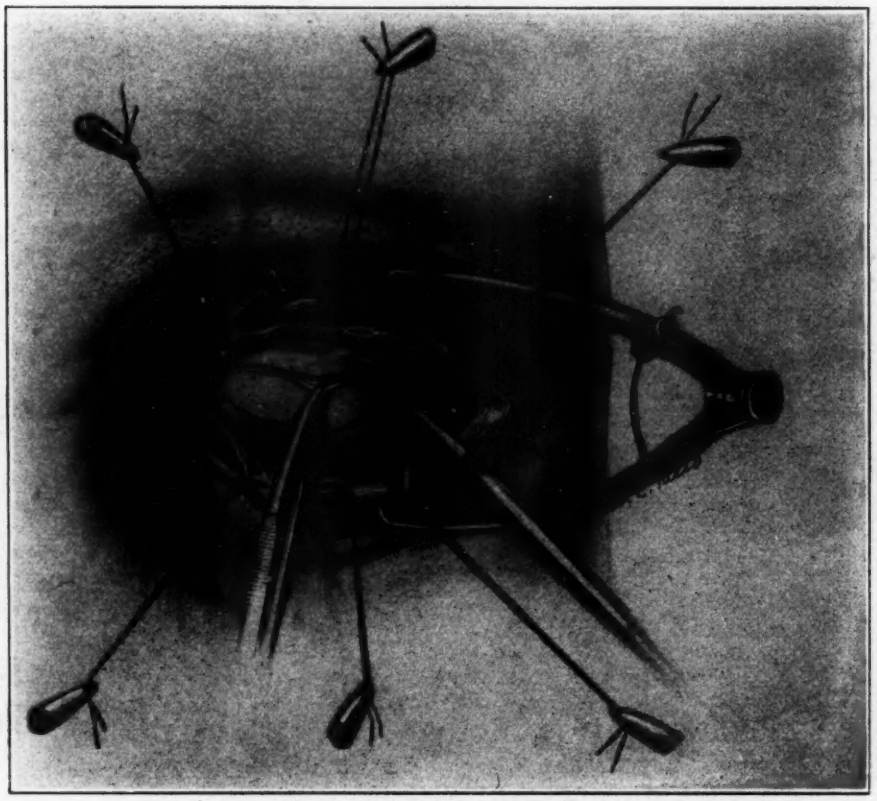

Fig. 2.—The scleral incision is deepened with a keratome.

Lindner's clinic and partial healing in several others. The case reported here, in which cure resulted, seems worth publishing.

REPORT OF A CASE

Mrs. E. H., aged 54, came to the clinic at the Stanford Medical School on Dec. 26, 1937, with the complaint that the vision of the right eye had suddenly failed a few weeks previously. The left eye had been blind since childhood. The patient had always been near sighted.

2. Ramach, F.: Arch. f. Ophth. **136**:78, 1936.

Examination of the right eye showed, briefly, a normal external eyeball. There were many opacities in the vitreous; the retina was detached and markedly elevated everywhere except nasally and above. There was a large typical horseshoe-shaped tear in the horizontal meridian, far temporally. The retina was atrophic in many places and did not move freely. Vision was limited to counting of fingers at 2 feet (61 cm.).

The iris of the left eye was atrophic, and a pinpoint pupil was filled with membrane. There was uncertain perception of light in this eye.

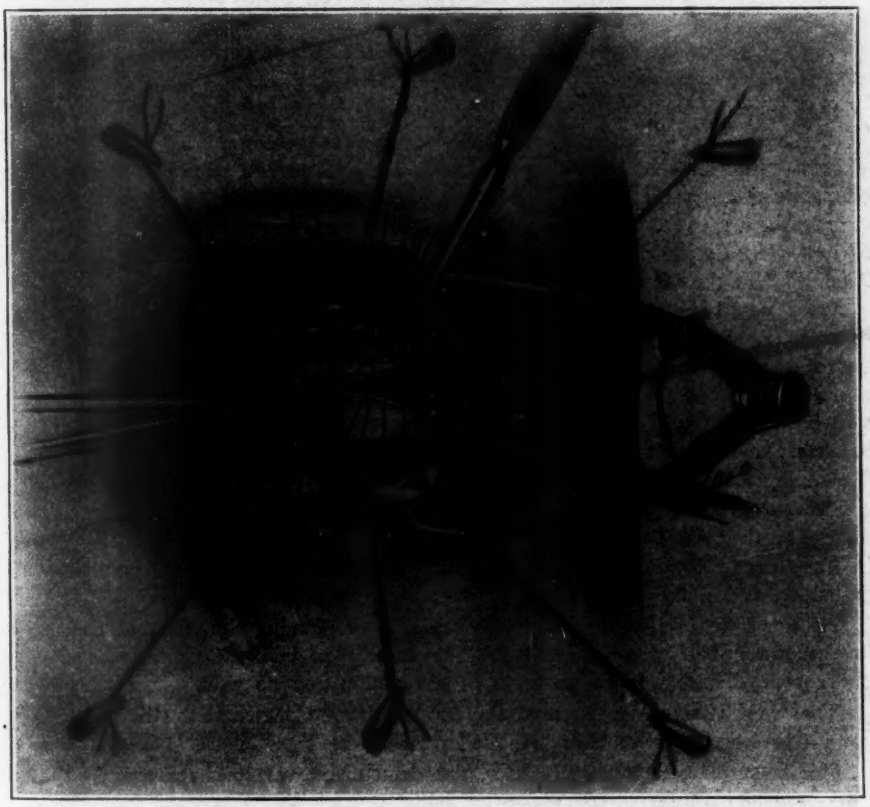

Fig. 3.—The scleral sutures have been put in place. The edges of the nasal portion of the scleral flap have been cut through with scissors and that part of the flap pulled under the untied sutures. The nasal sutures have been tied, the lips of the scleral wound being drawn together.

On December 29 a multiple diathermy puncture operation (Šafář) was performed. The position of the hole was verified by direct localization. Three Pischel pins were put in, and the location was found to be correct. The tear was then surrounded by a double row of pins 45 degrees above the horizontal and well below the hole and back 19 mm. from the limbus. The area inside the lines of pins was treated with surface coagulation by a Lacarrere electrode. Observation of the fundus showed good coagulation. On withdrawal of the pins (35 were used) much sticky fluid escaped. Convalescence was uneventful.

On Jan. 31, 1938, the patient was discharged with the retina in place. She could count fingers at 5 feet (162 cm.). The visual field was normal.

On February 18 the patient stated that five days before she saw flashes of light before the eye, and vision suddenly became poor. The retina was found to be attached above the horizontal meridian except at the tear, where it was detached. It was entirely detached below.

On March 1 the multiple diathermy puncture operation (Šafář) was again performed. A double row of pins was put in, again surrounding the hole, going from the edge of the superior rectus muscle to the edge of the inferior rectus muscle. The external rectus muscle was cut off. The Lacarrere electrode was used as before. Good coagulation was observed. After the pins were removed 1 cc. of air was injected into the vitreous cavity, after which considerable more fluid escaped. Convalescence was uneventful.

On March 20 detachment was still present below, although the tear seemed closed.

On April 12 the retina was detached below the horizontal meridian, where it stretched anteriorly in stiff white folds apparently to the ora serrata. About 2 disk diameters nasally and below the disk there was a peculiar puckering of the retina, almost like a rosette. Here the vessels dipped down into the center of the "rosette" as if it were the disk. Vision was limited to perception of fingers at 2 feet. The lower half of the visual field was present.

On June 24 the eyeball-shortening operation (Lindner) was carried out. This operation was done as described here, a scleral strip 2.5 mm. wide being taken out of the lower half of the globe, the strip extending from one lateral rectus muscle to the other and being 11 mm. from the limbus. The inferior rectus muscle was cut and was not resutured (monocular case). The first third of the scleral crescent was in the area operated on previously by the diathermy method. The many adhesions between the sclera and the choroid were carefully freed by a fine spatula in some places, and in others a thin membrane of scleral tissue was left. The denuded choroid was delicately touched with a cotton applicator dipped in 3 per cent solution of potassium hydroxide just before the scleral sutures were pulled up and tied.

At the close of the operation inspection of the fundus showed that the retina in the lower half was not stretched as tightly as before. Convalescence was uneventful. There was surprisingly little reaction and no pain.

Two weeks later inspection of the fundus showed detachment still present but not as elevated. The peculiar "rosette" was flatter.

On August 18, two months later, the detachment was perhaps a little flatter. There was no change in the vision or field.

On September 22, three months later, the detachment still was obvious but flatter. Vision improved from perception of fingers at 2 feet (61 cm.) to perception of fingers at 6 feet (183 cm.). There was a little restoration of the field above the horizontal.

On October 13, almost four months after operation, the retina was entirely back in place. The "rosette" had disappeared, and only a little gray area in the retina was seen at its former location. Vision was 11/200. The field was normal to a 5 mm. white test object at 330 cm.

The patient could get about by herself, but she still saw flashes of light, which were getting less frequent. She noted difficulty in seeing in the evening.

The patient was last seen on Aug. 30, 1939, fourteen months after operation. There was no change in her condition. Vision with a -1.00 sph. $\bigcirc$ -2.50 cyl., axis

25 was 15/100. With + 2.00 sph. ⊃ — 2.50 cyl., axis 25, she could read Jaeger's no. 7 test type and a few words of Jaeger's no. 6 test type.

It is interesting to note that the refraction of the eye was changed by the shortening operation from a — 4.50 sph. ⊃ — 0.50 cyl., axis 90 to a — 1.00 sph. ⊃ — 2.50 cyl., axis 25.

SUMMARY

A brief review of the eyeball-shortening operation (Lindner) is presented, with report of 1 case in which the procedure was used. It resulted in complete reattachment of the retina, with great improvement of vision and restoration of a normal visual field. The refraction of the eye was changed from a —4.50 sph.⊃—0.50 cyl., axis 90 to a —1.00 sph.⊃ — 2.50 cyl., axis 25, which gave vision of 15/100.

EFFECT OF ANOXEMIA ON THE DARK ADAPTATION OF THE NORMAL AND OF THE VITAMIN A–DEFICIENT SUBJECT

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AND
FRANCIS HEED ADLER, M.D.
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The tremendous increase in the sensitivity of the retina when it is kept in the dark for a prolonged period is common knowledge, and the quantitative measurement of this phenomenon is not new. It is also well known that certain pathologic conditions may affect this process of dark adaptation and give rise to varying degrees of night blindness. However, it has been only within the past decade that the knowledge of this fundamental principle has aroused widespread interest. The impetus was given by the realization that in certain metabolic disorders the course of the dark adaptation and final threshold (minimum light visible after a prolonged period in the dark) was altered.

These various metabolic disorders were linked to vitamin A deficiency by numerous investigators working in separate fields. The recognition of vitamin A as one of the products of decomposition of visual purple when acted on by light and its role in the resynthesis of visual purple in the dark showed rather conclusively that vitamin A was a factor in the chemical basis of vision.

There have been numerous reports on vitamin A deficiency and dark adaptation, but, as Hecht [1] has pointed out, they have been more enthusiastic than critical. Hecht and Wald [2] have both carried out experiments on vitamin A deficiency under well controlled conditions and have shown that both the rods and the cones are affected in the deficient state. The fact that both thresholds are raised shows that vitamin A must be con-

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Supported by a grant from the John and Mary Markle Foundation.

From the Department of Ophthalmology and the Johnson Foundation for Medical Physics, University of Pennsylvania.

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 7, 1939.

1. Hecht, S.: The Relation Between Vitamin A and Dark Adaptation, J. A. M. A. **112**:1910 (May 13) 1939.

2. Wald, G.; Jeghers, H., and Arminio, J.: An Experiment in Human Dietary Night-Blindness, Am. J. Physiol. **123**:732 (Sept.) 1938.

cerned with the chemical cycle of both the rod and the cone vision. Wald [3] has isolated iodopsin, or visual violet, from the cones, which is apparently analogous to the rhodopsin, or visual purple, of the rods.

There are other metabolic functions which are directly concerned with the visual process, one of the most important being the adequate supply of oxygen in the circulating blood. The effect of anoxemia has been extensively studied, and its physiologic and psychologic manifestations are well known. Wilmer and Berens [4] over twenty years ago reported on the effects of anoxemia on visual acuity, accommodation, muscle imbalance and the visual field. Of the vast amount of literature on the subject, that of McFarland is especially noteworthy, since he has reported with Knehr and Berens [5] and with Evans [6] on the effects of anoxemia on various visual functions.

Hecht and his associates have done a great deal to elucidate the normal physiologic function of the retina, and since the effect of anoxemia has been fairly thoroughly investigated, we felt that an attempt should be made to correlate and clarify its probable site of action on the visual response. Measurement of the light sense during the course of dark adaptation is probably the best means at one's disposal for investigating the fundamental basis of the visual response.

EXPERIMENTAL PROCEDURE AND RESULTS

The measurement of dark adaptation presupposes previous light adaptation. This is followed by brief exposures to a flash of light the intensity of which is adjusted so that it is just visible. The thresholds so determined are usually plotted in logarithmic units of intensity, since an increase in sensitivity of 1 to 100,000, which is not uncommon, if plotted in the ordinary way would mask the true course of dark adaptation. We used the adaptometer devised by Hecht and Shlaer [7] and the procedure recommended by them throughout these experiments.

The normal dark adaptation curve of our subject is shown in chart 1. The first part of the curve is the adaptation curve of the cones when the test flash appears colored, and the plateau is known as the final cone threshold. Cone function can be studied separately by using a cen-

3. Wald, G.: Photo-Labile Pigments of the Chicken Retina, Nature, London **140**:545 (Sept. 25) 1937.

4. Wilmer, W. H., and Berens, C.: Medical Studies in Aviation: V. The Effect of Altitude on Ocular Functions, J. A. M. A. **71**:1394 (Oct. 26) 1918.

5. McFarland, R. A.; Knehr, C. A., and Berens, C.: The Effects of Anoxemia on Ocular Movements While Reading, Am. J. Ophth. **20**:1204 (Dec.) 1937.

6. Evans, J. N., and McFarland, R. A.: The Effects of Oxygen Deprivation on the Central Visual Field, Am. J. Ophth. **21**:968 (Sept.) 1938.

7. Hecht, S., and Shlaer, S.: An Adaptometer for Measuring Human Dark Adaptation, J. Optic. Soc. America **28**:269 (July) 1938.

trally fixated test flash subtending a retinal angle of 2 degrees which falls on the rod-free fovea or by using a deep red light ($\lambda = 670$ millimicrons) to which wavelength the rods are relatively insensitive. The second part of the curve represents the adaptation of the rods when the test flash appears colorless. The final plateau is the final rod threshold.

The subject, E. M., a healthy well trained observer, had been the subject for dark adaptation experiments for several months prior to the present investigation. Anoxemia was induced by having him breathe a gaseous mixture of 50 per cent air and 50 per cent nitrogen which contained approximately 10.4 per cent (by volume) of oxygen (normal, 20.9 per cent).[8] This was supplied from two large spirometers, and a special mouthpiece was devised so as not to interfere with

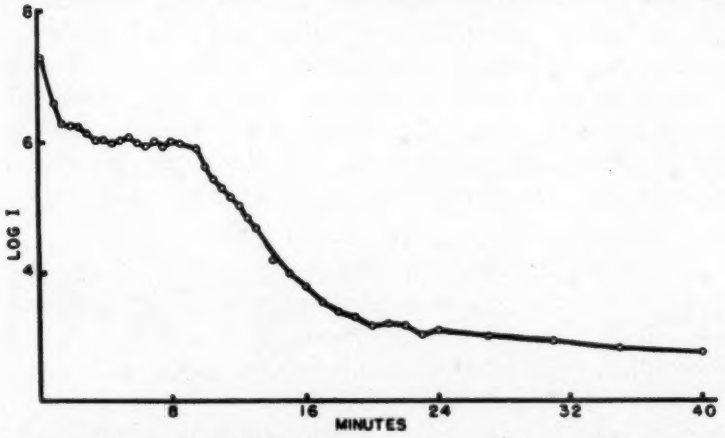

Chart 1.—Normal dark adaptation curve of observer's right eye. The preadapting intensity was 4,900 millilamberts, and the time of exposure, three minutes. The unit of threshold intensity is the micromicrolambert and is the same for all experiments. The test flash was exposed for one-fifth second.

testing the visual response. Several control experiments showed that the added apparatus did not interfere with the visual test.

The effect of anoxemia was studied first on the cone and rod thresholds. The cone threshold was studied by inserting a red filter in the beam of the test flash, and, as chart 2 will show, only the cone part of the adaptation curve was recorded. The effect of anoxemia was to raise the threshold by a factor of 2.5 (an upward displacement of 0.4 units in the intensity plot on the logarithmic scale). To test the effect on the rod threshold, we allowed the subject to become completely dark adapted before we induced oxygen lack, and again the threshold

8. Analysis of gas at different times during the series of experiments showed that the oxygen content varied from 9.8 to 10.4 per cent (by volume).

was raised by a factor of 2.5 (chart 3). Our apparatus did not permit the study of anoxemia on the entire curve, as the capacity of the spirometers was sufficient to last only for fifteen to twenty minutes. In order to test the rate of adaptation we reduced the preadapting light to one tenth of the usual intensity; with this preadapting intensity the final rod threshold is reached within twenty to twenty-five minutes.

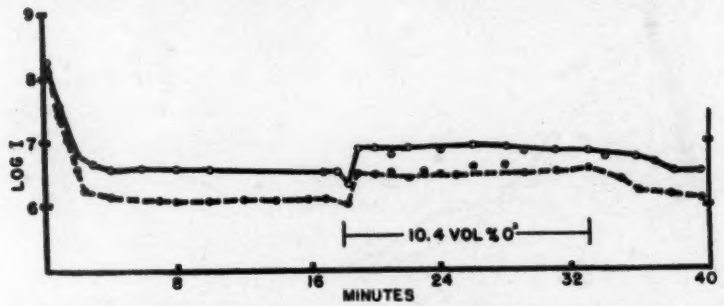

Chart 2.—Dark adaptation curves of subject's right eye showing the final cone threshold with the use of a red filter (λ — 680 millimicrons). The preadapting intensity was 4,900 millilamberts. The normal curve and the effect of anoxemia are indicated by the solid circles and the effect of vitamin A deficiency and anoxemia by the open circles.

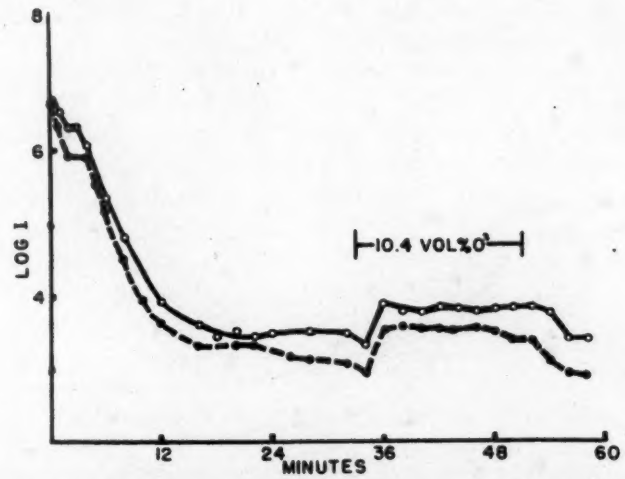

Chart 3.—Dark adaptation curves with a preadapting intensity of 510 millilamberts, showing the effect of anoxemia on the final rod threshold. The normal curve and the effect of anoxemia are indicated by the solid circles and the effect of vitamin A deficiency and anoxemia by the open circles.

Chart 4 shows that the effect of anoxemia is simply to raise the entire curve on the intensity scale and that the rate of adaptation is approximately the same.

The subject was then placed on a diet deficient in vitamin A which was calculated to supply him with 250 to 350 Sherman units per day. His diet was supplemented with the estimated requirement of the other vitamins in concentrated form.[9] The rise in threshold due to vitamin A

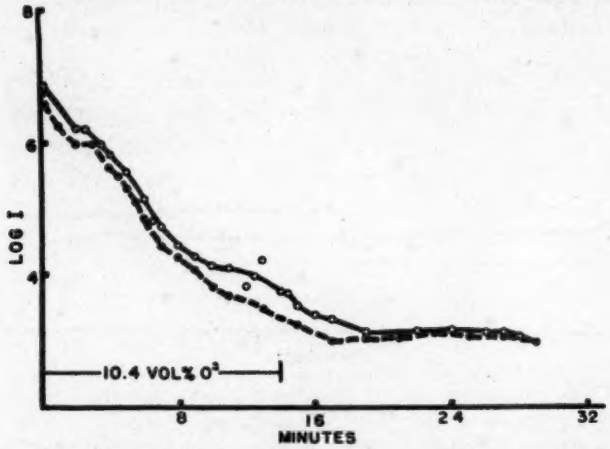

Chart 4.—Dark adaptation curves with a preadapting intensity of 510 millilamberts. The normal curve (no oxygen lack) is indicated by the solid circles and the effect of oxygen lack, which was induced one and one-half minutes before the preadapting light was turned off, by the open circles. The subject was not deficient in vitamin A. The effect of vitamin A deficiency was simply to raise both curves on the intensity scale.

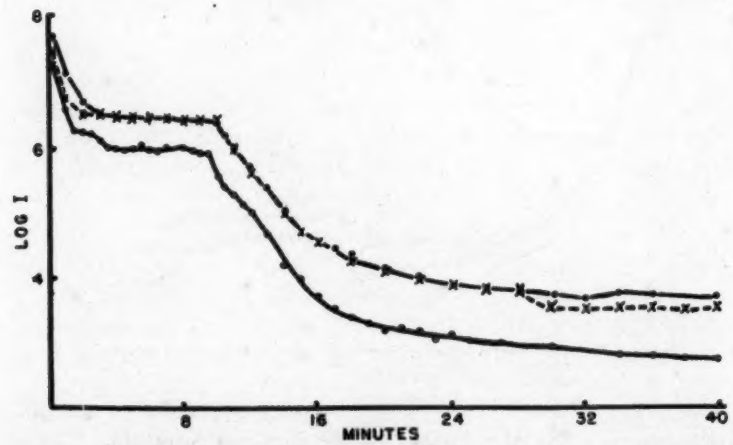

Chart 5.—Change in the dark adaptation curve due to vitamin A deficiency. The preadapting intensity was 4,900 millilamberts. The normal curve is indicated by the open circles; the curve plotted the fourth week of the deficient diet, by the crosses, and the curve plotted the sixth week of the deficient diet, by the solid circles.

9. Eli Lilly & Co. supplied the various vitamin concentrates used throughout these experiments, and the S.M.A. Corporation, the pure vitamin A.

deficiency was first noticeable on the fourth day after the dietary restriction. The rise continued until about the fifth week; and then the level remained practically constant until the conclusion of the experiment in the ninth week. Chart 5 shows the change that occurs in the dark adaptation curve in experimental vitamin A deficiency. It will be noted that there is an unequal rise in the two plateaus, the rods showing the greater rise.[10]

When the rise in threshold due to the vitamin A deficiency was well marked, anoxemia was again produced in the subject as a result of breathing a mixture containing 10.4 per cent (by volume) of oxygen. There was no appreciable change in the effect of anoxemia when he was tested at various times during the period of his dietary restriction. This will be evident by looking at the upper curves of charts 2 and 3, which show a fairly marked rise in threshold due to the vitamin A deficiency and a still further rise in the threshold, by a factor of 2.5 (log I = 0.4), due to anoxemia. The rate of adaptation when tested was also unchanged.

COMMENT

The normal dark adaptation curve was altered by a change in certain fundamental metabolic requirements. We first reduced the amount of oxygen in the circulating blood and during the period of anoxemia found a temporary but significant upward displacement of the intensity curve by a factor of 2.5 (log I = 0.4); that is, the rod and cone thresholds were both raised by an equal amount, but the rate of adaptation was apparently unchanged. We then reduced the dietary intake of vitamin A and changed the character of the adaptation curve again. This change, however, caused an unequal rise in the rod and cone thresholds, the rod threshold showing the greater variation. This would suggest that even though anoxemia and vitamin A deficiency both change the character of the adaptation curve, they act on different processes in the visual cycle. The rise in threshold due to anoxemia is simply additive and is the same in the normal and in the vitamin A–deficiency state.

10. The fact that the threshold did not continue to rise after the fifth week may be due to the fact that our subject was receiving 250 to 350 units of vitamin A per day in his prescribed diet, whereas both Hecht's and Wald's experimental subjects did not receive more than 200 units per day. A few days before the subject was returned to his normal diet he was given two large doses of vitamin A concentrate by mouth (50,000 and 100,000 U. S. P. units). We did not find any precipitous drop in the threshold as noted by Wald. After two weeks of a diet high in vitamin A but without any added concentrates, our subject's threshold levels were practically as high as at the peak of his deficiency. He was then given 50,000 units (U. S. P.) of vitamin A in gelatin capsules per day. There was no appreciable improvement at the end of one week.

The unequal rise of the rod and cone threshold associated with experimentally induced vitamin A deficiency has been previously noted by Hecht and by Wald. Fischer and Jongbloed,[11] Bunge [12] and McFarland and Evans [13] have reported on the effects of anoxemia on dark adaptation. Fischer and Jongbloed stated that the rate of adaptation is delayed and attributed this to a delay in the regeneration of visual purple. However, if the threshold is raised due to any cause, it will naturally take longer to reach a certain level on the intensity scale, but the rate need not be changed. Reference to chart 4 will show that during the period of anoxemia both the initial and the final threshold are raised, but the slope of the two curves is practically the same; this would indicate that the rate of regeneration of visual purple is unaffected. We would attribute the effect of anoxemia to other than the photochemical response.

The effect of oxygen lack in the excised nerve has been studied by Lehmann [14] and others. He found that the threshold to stimulation was first lowered and then rose rapidly. This is mentioned, as in several of our experiments we found a slight drop in the threshold if the test flash was exposed within the first minute after the induction of anoxemia. Hartline [15] has also shown in his experiments on single nerve fibers in the frog's retina that complete anoxia causes a cessation of the response of the optic nerve, which will recover if the period of oxygen lack is not of too long duration.

In considering the effect of anoxemia, one must consider the fact that these experiments were carried out under normal atmospheric pressure and that the anoxemia was induced rapidly and was of short duration. The fact that one could reproduce the same change in threshold from day to day and that there was apparently no mental confusion on the part of the observer, as tested by questioning and by taking samples of his handwriting, is fairly good evidence that this was not a gross disturbance of intelligence or coordinated behavior on the part of the observer. The observer was twice subjected to audiometer tests

11. Fischer, F. P., and Jongbloed, J.: Untersuchungen über die Dunkeladaptation bei herabgesetztem Sauerstoffdruck der Atmungsluft, Arch. f. Augenh. **109**: 452, 1936.

12. Bunge, E.: Verlauf der Dunkeladaptation bei Sauerstoffmangel, Arch. f. Augenh. **110**:189, 1937.

13. McFarland, R. A., and Evans, J. N.: Alterations in Dark Adaptation Under Reduced Oxygen Tension, Am. J. Physiol. **127**:37 (Aug.) 1939.

14. Lehmann, J. E.: The Effect of Asphyxia on Mammalian A Nerve Fibers, Am. J. Physiol. **119**:111 (May) 1937.

15. Hartline, H. K.: The Response of Single Optic Nerve Fibers of the Vertebrate Eye to Illumination of the Retina, Am. J. Physiol. **121**:400 (Feb.) 1938.

(with both a 2-A and a 4-A Western Electric audiometer) during periods of anoxemia, and no change from his normal hearing was demonstrable.

CONCLUSIONS

The normal character of the dark adaptation curve has been altered by a change in two metabolic requirements. The changes produced by anoxemia and vitamin A deficiency are dissimilar, and this suggests that two different processes in the visual response are involved. This is further substantiated by the fact that vitamin A deficiency does not alter the effect produced by oxygen lack. It is known that vitamin A is concerned with the photochemical basis of the visual response, and it is probable that anoxemia acts elsewhere in the visual system, i. e., on the nerve mechanism.

DISCUSSION

Dr. Francis H. Adler, Philadelphia: As a result of our experiences with measuring dark adaptation, we should like to issue a word of caution in the examination of patients with the instruments which are now on the market. It is obvious that the type of work which Dr. McDonald has reported necessitates a carefully controlled apparatus and an intelligent, able observer. We can say definitely when patients are examined with the Hecht apparatus whether their rate of adaptation or their final threshold is normal; in other words, we can say whether they do not have a vitamin A deficiency, but when one has a patient whose threshold is raised above what one normally sees, one must use a good deal of caution in saying that that is due to vitamin A deficiency. The patient may be a poor observer and therefore not a good subject for testing. He may have some other deficiency in his retina other than a vitamin A deficiency which accounts for the raised threshold. We have seen numerous persons with various types of retinal degeneration in whom we believe this to be the case, or they may actually have a vitamin A deficiency; however, with the apparatus which is on the market today one must exercise a good deal of caution in interpreting small changes in the rate of adaptation as due to vitamin A deficiency. We believe that there is no evidence that any considerable proportion of the population is suffering from vitamin A deficiency such as has been reported in the literature. There is good evidence that a normal person whose rate of adaptation is normal cannot have that adaptation improved by taking vitamin A. There is no necessity, therefore for a normal person to take tablets containing vitamin A when driving at night.

Dr. Henry P. Wagener, Rochester, Minn.: In line with what Dr. Adler has just said, I want to call attention to some work which has recently been done by Steffen for his master's degree in the Mayo Foundation of the University of Minnesota, along with Bair and Sheard. They have been able to state that in an otherwise normal person reduction of vitamin A in the diet is certainly not necessarily followed by a reduction of light sensitivity or a reduction in dark adaptation. In 3 cases in which the subjects were placed on a diet supposedly free from vitamin A for one hundred and sixty days as well as could

be determined by the dietitians, there was at no time during this period a definite decrease in light sensitivity which was any greater than could be observed during a similar period in which they were on a diet high in vitamin A. During that period, however, there did develop evidence in the ectodermal tissues of the skin which apparently was regarded by dermatologists as indicating vitamin A deficiency, so that it might be assumed that a certain degree of deficiency of vitamin A really developed, but during this period of time we were unable to demonstrate any consistent rise in the light threshold.

DR. ROBB MCDONALD: I should like to give a word of warning concerning these experiments on vitamin A deficiency. It is still not known that the changes are reversible. Recently I tested a patient who had been off the vitamin A–deficient diet for three weeks—I am sure he was deficient with the Hecht apparatus—with a normal diet but high in vitamin A; his threshold was not normal after two weeks. The third week I gave him 50,000 units of vitamin A per day by mouth and some carotene intramuscularly, and his threshold is still as high as it was at the highest point in the deficient stage. I do not have any doubt that I can cure him, but I think a word of warning should be given concerning experimental vitamin A deficiency until it is known that the changes are reversible.

DR. PARK LEWIS, Buffalo: If the liver is the storehouse of vitamin A, is it known how long that reserve supply endures? It might be that with this stored supply there would be no manifestations in the eyes so long as that could be drawn on. I should like to know whether that enters into the question.

DR. ROBB MCDONALD, Philadelphia: I think it does, but Hecht, who has done excellent work at Columbia University on vitamin A deficiency, was able to show during a control period (before the diet was started he gave large doses of vitamin A) that within two or three days he was able to pick up some deficiency in the final rod threshold. The cone deficiency does not show up before five days. I feel that at the end of four days I was able to pick up a noticeable change. The observer was used for six months before I put him on the diet, and he was very accurate. Even if the liver has a large store of vitamin A, I think one can pick up early signs of deficiency by testing the light sense in the eye, but, as Dr. Adler pointed out, impairment of light sense does not necessarily infer vitamin A deficiency.

MALIGNANT MELANOMA—SO-CALLED SARCOMA—OF UVEA

II. PROBLEMS IN DIAGNOSIS

T. L. TERRY, M.D.
BOSTON

Mistakes in the diagnosis of malignant melanoma [1] are numerous. They are of two general types, namely: (1) failure to discover the presence of the tumor and (2) confusion of some other pathologic condition with malignant melanoma.

FAILURE TO DISCOVER THE PRESENCE OF THE TUMOR

Failure to discover the presence of the tumor appears to be the more common error. In a recent statistical study of 94 cases of tumor [2] it was found that 42 of the eyes were enucleated without the surgeon's suspecting that a neoplasm was in the eye. The correct diagnosis was established in the pathologic laboratory. The presence of the tumor always was obscured by some more obvious but usually closely related clinical entity (table 1). The fundus in many instances could not be examined ophthalmoscopically.

Glaucoma, a common complication of the neoplasm, was found in 41 of 94 cases (44 per cent). The tumors usually were not diagnosed because they could not be seen. In 24 cases the fundus was not visible, and in the remaining instances the tumors were in front of the equator. When glaucoma is suspected, the completeness of examination of the fundus, especially in the periphery, is limited by justifiable unwillingness to use a mydriatic. The institution of miotic therapy limits the study to an even smaller area. It is obvious, then, that intraocular neoplasm should be suspected in every case of unilateral glaucoma. Although there was no example of bilateral glaucoma, it should be borne in mind that malignant melanoma doubtless can exist even if bilateral glaucoma

From the Harvard Medical School and the Massachusetts Eye and Ear Infirmary.

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

1. Since the origin of this neoplasm may not be from mesodermal tissue, it is best to avoid the term sarcoma.

2. Terry, T. L., and Johns, J. P.: Am. J. Ophth. **18**:903 (Oct.) 1935.

is present. The fact that glaucoma most commonly hides malignant melanoma is sufficient argument against any palliative operation for glaucoma in a blind eye.

Separation of the retina, being present in 85 of 94 cases (90 per cent),[2] is the most common complication of malignant melanoma of the choroid. The separation may not be evident clinically, but, on the other hand, it may be extremely large and markedly out of proportion to the size of the neoplasm. The retinal separation associated with neoplasm appears to be due to a subretinal exudate derived from the altered choroid or from the tumor. The retina is usually adherent to the apex of the growth, and the separation of the retina occurs almost invariably around the neck of a pedunculated tumor. The retina is not long enough to cover the increased surface produced by the pedunculated growth of the tumor. The subretinal fluid in malignant melanoma is viscous and only

TABLE 1.—*Erroneous Clinical Diagnoses in Ninety-Four Cases of Malignant Melanoma of the Choroid*

Clinical Diagnosis	No. of Cases
Glaucoma	31
Separation of the retina	4
Iridocyclitis	1
Panophthalmitis	1
Disorganized globe	2
Infected perforating wound	1
Ulcerative keratitis	1
Intraocular hemorrhage	1
Total	42
Phthisis bulbi (2 cases not reported in this series)	

semifluid, whereas the subretinal fluid in simple separation of the retina appears relatively thin, resembling fluid vitreous.[3] The subretinal fluid associated with neoplastic separation does not flow rapidly through openings in the sclera and can be removed only with difficulty through a hypodermic needle of large gage, since it tends to coagulate on exposure to air. The tumor and the very viscous exudate, if large in amount, often produce an abnormally heavy eye, the eye being increased in weight sufficiently to be obvious when held in the hand.[4] The exudate

3. According to Franta (Ceskoslov. oftal. 3:201, 1937), the percentage of albumin in subretinal fluid under a simple separation of the retina increased with the age of the separation from 0.770 to 3.345.

4. The weight of the relatively normal eye is from 6.7 to 7.2 Gm., as determined by weighing a series of 147 eyes. Eyes unusually large from myopia or buphthalmos may be increased in weight up to 9 Gm. Eyes of relatively normal size weighing over 7.2 Gm. were found to contain hemorrhage, organized exudate, heavy coagulated subretinal exudate or malignant melanoma of the choroid, the last two conditions being the most frequent. Several eyes containing small tumors weighed well within the average.

coagulates solidly in a short time after fixation of the enucleated eye in a solution of formaldehyde. The subretinal fluid associated with a neoplasm, therefore, is not likely to run out through openings made by the Šafář or the Gonin operation.

Holes, rarely present in the separation of the retina secondary to malignant melanoma, are usually near the ora serrata.[5] It is presumed that the hole is the result of tension produced in some cases of pedunculated tumor first from adherence of the retina to the apex of the tumor and later from the growth of the tumor away from the ora serrata.

In contrast to simple separation, the retinal separation secondary to the neoplasm is not tremulous on movement of the eye or on pressure on the eye. Separation of the retina secondary to a choroidal neoplasm does not tend to shift its location because of gravity. More than one distinct separation of the retina can be present in association with a

Fig. 1.—*A* (case 1), scar tissue, resulting from a Šafář operation, firmly attached to the sclera in the region of the tumor. *B* (case 2), thickening of the sclera by fibrosis in the region of the Šafář operation. The tumor has spread around the optic nerve head and can be seen on the side of the eye opposite the main body of the neoplasm. Near the nerve head, on the side of the main mass, an extension of the malignant melanoma can be seen in an emissarium. (Figure 4 shows this area under high power magnification.)

tumor. In a case recently observed there were two unrelated distinct separations, one in the region of the neoplasm just above the nerve head and the other at a considerable distance below the nerve head.

Two eyes (cases 1 and 2 and figs. 1 to 4) containing malignant melanomas have been studied on which the Šafář operation was performed to reattach the separated retina. The amount of the separation was not reduced by the operation, nor was any subretinal fluid obtained through the puncture wounds. Finally, the establishment of the correct diagnosis led to enucleation. With one exception, the small operative

5. Wintersteiner, H.: Die Geschwülste des Uvealtraktus, Ergebn. d. allg. Path. u. path. Anat. (supp.) **10**:1044, 1907.

wounds through the sclera, made by Walker pins, were filled by means of scar tissue formation. It is possible that the scars may have formed more rapidly than the tumor cells grew, filling the wounds with tissue relatively impervious to the advancement of the tumor. On the other hand, tumor cells that may have grown into the operative wounds soon after operation were destroyed by the advancement and contraction of the scar tissue. However, in one wound in case 2 the tumor had extended one third of the way through the sclera. In this case the neoplasm had

Fig. 2 (case 1).—Scar tissue in the sclera filling the operative wound made during the second Šafář operation. Although the scar tissue was relatively loose, it had pushed out or destroyed any tumor that might have penetrated into the wound.

penetrated deeply into the sclera along an emissarium some distance from the field of operation. The findings in these 2 cases by no means prove that the neoplasm cannot penetrate entirely through a small puncture wound of the sclera. Tumors have been observed to grow out through so-called diagnostic punctures [6] and through operative wounds.[5]

6. Scheerer, in discussion on Rönne, H.: Diagnosis of Choroidal Melanosarcoma, Arch. Ophth. 1:397 (Jan.) 1929.

It is just as important to suspect the possibility of intraocular neoplasm in each instance of separation of the retina as in each case of glaucoma.

Uveitis, another common complication, evident in 75 of 94 cases of malignant melanoma,[2] often is not severe, being demonstrable only on microscopic study of the eye. In some instances, however, it may be severe and affect the eye so extensively that the choroidal tumor is

Fig. 3 (case 2).—Scar tissue along the tract made through the sclera with a Walker point. In the upper part of the illustration the tumor is seen within the eye. It had penetrated into the wound and can be seen incarcerated in the scar tissue. The tumor looks pigmented, but the difference in color from sclera is caused by difference in staining properties of the scleral scar and the tumor.

hidden. Toxins from inflammation and necrosis within the tumor itself may be responsible for the uveitis, although severe uveitis frequently is seen with long-standing simple separation of the retina. The cases of iridocyclitis and panophthalmitis in table 1 are examples of cases in which severe uveitis was present due to tumor. Case 3 is a second

example and Cohen's [7] case is a third. In Cohen's case the malignant melanoma spread rapidly through the orbit after evisceration of the globe. It is argued in general that evisceration of the globe is preferable to enucleation in the face of purulent endophthalmitis and panophthalmitis because of fear of extension of the intraocular infection to the meninges along the intervaginal space laid open by the enucleation. Verhoeff [8] expressed the belief that evisceration should never be done except in unusual circumstances, such as immediately after an expulsive hemorrhage complicating cataract extraction. He pointed out that evisceration of the globe in the presence of endophthalmitis and panophthalmitis leaves a puckered collapsed sclera, which prevents good drainage, a condition likely to produce meningitis.

Fig. 4 (case 2).—Extension of the tumor along an emissarium deeply into the sclera. A posterior ciliary artery can be seen surrounded by the neoplasm. The location and the direction by which this extension has grown are proof that it is not following the tract of an operative wound.

Since disorganized globes and even shrunken globes [9] occasionally may contain malignant melanomas, it is greater wisdom to remove these useless eyes than to cover them with a prosthesis, which may well be tolerated because of decreased or absent corneal sensitivity.

In a recent communication Dr. Frank E. Burck stated that he had found recently for the third time in his experience a malignant melanoma in a shrunken globe. In his last case, one in which an eye was eviscerated

7. Cohen, M.: Tr. Am. Ophth. Soc. **30**:96, 1932.
8. Verhoeff, F. H.: Personal communications to the author.
9. Chance, B.: Am. J. Ophth. **17**:48, 1934.

in which sight had been lost for ten years and phthisis had developed, complete removal of the uvea led to a suspicion of its malignant character, which was verified by quick section of the eviscerated material.

Likewise, blind eyes with infected wounds, destructive injuries, ulcerative keratitis (see case 4 and figure 6) and intraocular hemorrhage, in short all painful or inflamed blind eyes, should be removed by enucleation if patients are to be given the proper protection against

Fig. 5 (case 4).—Extensive hypopyon ulcer of the cornea in an eye containing a malignant melanoma of the choroid. The hypopyon was largely lost or dissipated during fixation and preparation of sections.

malignant neoplasms of the choroid, a condition which is fatal to 41 per cent.[2]

CONFUSION OF SOME OTHER PATHOLOGIC CONDITION WITH MALIGNANT MELANOMA

Confusion of some other pathologic condition with malignant melanoma of the choroid is a less frequent error. Mistakes of this type no doubt will continue to occur. In so far as life is concerned, failure

to remove an eye containing a malignant melanoma is a greater mistake than removal of an eye erroneously thought to contain the neoplasm. Frequently the pathologic lesion clinically resembling the neoplasm destroys vision in the affected eye. A needless sacrifice of the globe may rob the patient of considerable vision and even possibly of his only eye, because some lesions resembling the malignant melanoma clinically may tend to be bilateral, may heal with considerable resolution or may affect visual efficiency little.

As shown by specific examples, a number of conditions have been mistaken for malignant melanoma:

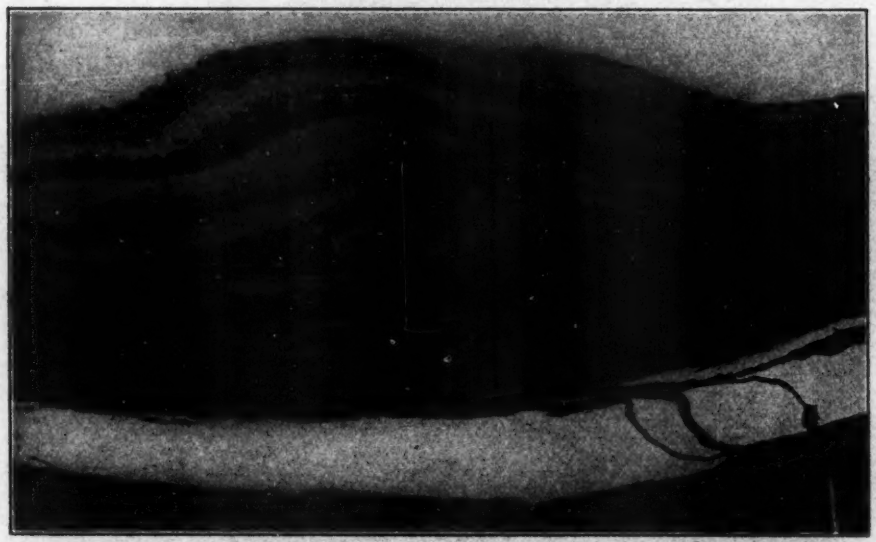

Fig. 6 (case 5).—Disciform degeneration of the macula. Organized hemorrhage beneath the pigment epithelium of the retina is the essential lesion. Note the subjacent nodule of chronic inflammation in the choroid.

Disciform degeneration of the macula
Localized inflammatory tumors of the choroid
Spontaneous separation of the choroid
Subchoroidal hemorrhage
Localized melanosis oculi
Angioma
Metastatic carcinoma
Metastatic chorioepithelioma
Retinoblastoma
Neurofibroma

At least seven eyes have been enucleated because disciform degeneration of the macula was mistaken for malignant melanoma. Verhoeff

and Grossman [10] reported 2 of these cases and also cited references to 4 in the literature. An additional instance is case 5 in this paper (fig. 6). The condition is often bilateral, 133 eyes being involved in 88 persons. Although hemorrhages in the retina and under the retina were observed with some frequency, usually the picture is that of a gray-green or almost black mound, which, except for pigment deposits, becomes white in time. Elevation of the lesion often reached 6 diopters, and at least some of the edge stood out sharply. Central dimpling was observed in some cases. The location almost invariably has been between the

Fig. 7.—Large flat inflammatory nodule of the choroid, clinically simulating a malignant melanoma.

superior and the inferior temporal branches of the central retinal artery. Circinate retinitis was present in a number of cases.

There are two general types of disciform degeneration, commonly called juvenile and senile. The juvenile type tends to occur in persons of less than 40 years of age, although Verhoeff and Grossman [10] considered the eye of a 63 year old patient (case 3) to be affected with this type. The juvenile type tends to heal, since in all probability it represents only a serous exudate between the pigment epithelium of the retina and the lamina vitrea. The other eye, possibly free at first from

10. Verhoeff, F. H., and Grossman, H. P.: Pathogenesis of Disciform Degeneration of the Macula, Arch. Ophth. 18:561 (Oct.) 1937.

ophthalmoscopic evidence of pathologic involvement, might be involved with disciform degeneration at a later date with ultimately more disastrous results to vision.

The senile type of disciform degeneration, occurring usually but not invariably after 40 years of age, is caused by hemorrhagic extravasations between the pigment epithelium and the lamina vitrea. Although the macular vision is destroyed permanently, the lesion does not lead invariably to complete loss of vision, a relatively good peripheral field persisting.

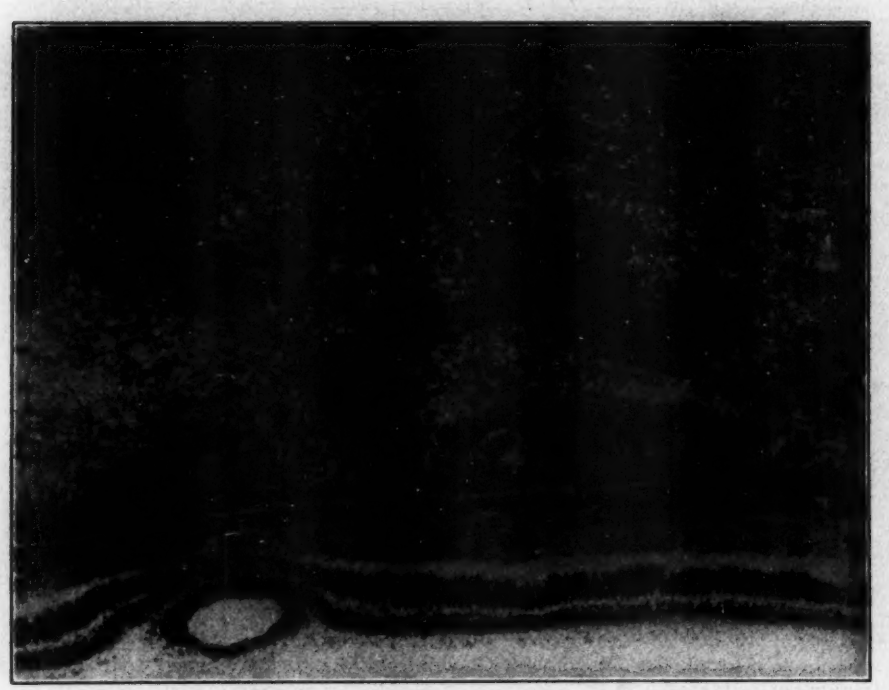

Fig. 8.—Higher magnification of an inflammatory nodule of the choroid showing many subretinal and choroidal giant cells.

Differential diagnosis between malignant melanoma and disciform degeneration is extremely important. Since disciform degeneration tends to be bilateral, the eye which may have given the better ultimate vision of the two sometimes is enucleated mistakenly for neoplasm. More particularly is this true in the juvenile type, in which recovery and good vision are not entirely unknown. Differentiation is possible in some instances only by waiting to observe developments. Not only should one study the fundus and make records, preferably by photography of the fundus, but one should also record the visual fields carefully. No increase in the size of the scotoma in a few weeks or a

few months indicates strongly that the lesion is not a malignant melanoma. Even decrease in the size of the scotoma may be observed.

Localized inflammatory lesions of the choroid productive of an enlargement and thickening of the part can simulate a malignant melanoma of the choroid most closely. However, many lesions of this type cause no real diagnostic trouble because of the frequency with which they extend into the retina by continuity, giving rise to pigmentary invasion and the production of a white scar. In these instances opacities of the vitreous are almost invariably present. Recently Gundersen brought to my attention a case [11] of his, which he has permitted me to summarize. He found a tumor in the right eye in the upper nasal portion of the fundus of a woman 31 years of age. The tumor was

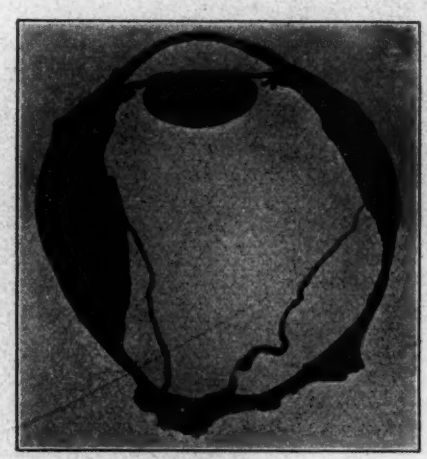

Fig. 9 (case 6).—Extensive separation of the choroid associated with severe iridocyclitis.

raised some 8 diopters. Drs. F. H. Verhoeff, J. Herbert Waite, Sanford Gifford and Paul Chandler also saw the patient. The consensus was that the lesion represented a malignant melanoma of the choroid. The eye was enucleated. The lesion proved to be entirely inflammatory (figs. 7 and 8). In the choroid considerable fibrosis, many giant cells and a large number of polymorphonuclear leukocytes were present. Corresponding to the area of choroiditis was a flat separation of the retina. The fact that an inflammation of the choroid, probably tuberculous, has simulated malignant melanoma clinically is the important point of the case for this paper.

11. This unique and important case warrants a more detailed consideration than could be given properly in this paper. It will be reported fully by Gundersen at a later date.

It is most improbable that a postoperative separation of the choroid would be mistaken for a malignant melanoma, since it is so commonly observed during convalescence after intraocular operations. However, spontaneous separation of the choroid, likely to be confusing, particularly when associated with a subretinal exudate and secondary to severe uveitis, can present a real problem if one bears in mind that a nonpigmented tumor may be transilluminated (as will be mentioned later). Spontaneous separation of the choroid is likely to give rise to a deep

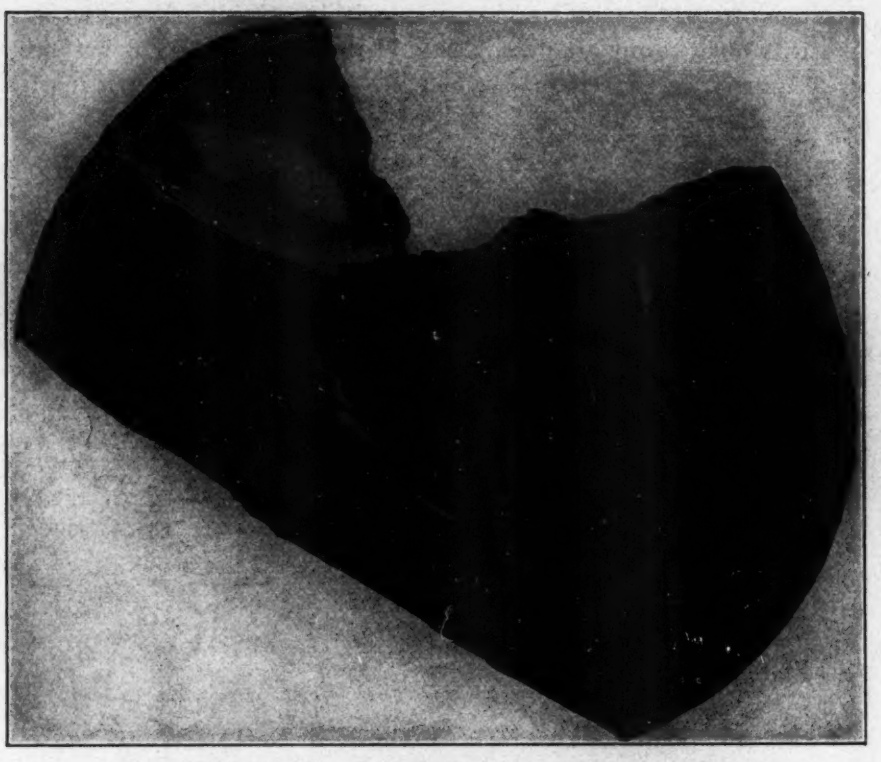

Fig. 10 (case 7).—Organized subchoroidal hemorrhage.

anterior chamber and a soft eye. Such an instance is represented in case 6 (fig. 9).

An old organized subchoroidal hemorrhage of considerable size simulated malignant melanoma sufficiently to result in enucleation in case 7 (fig. 10). The cause of the subchoroidal hemorrhage is not obvious from the history or the pathologic specimen. It must have recurred several times in the eye, since only a small quantity of hemorrhage in an unperforated eye would raise the intraocular pressure to approach the systolic arterial pressure. The increased intraocular pressure would stop the hemorrhage temporarily, at least until sufficient time elapsed for a quantity of aqueous to escape.

Doherty [12] pointed out that 25 per cent of patients with melanosis ultimately show malignant tumors of the choroid and that melanosis oculi may be localized. In a case of localized melanosis oculi, different from a benign circumscribed melanoma, the condition did simulate a malignant melanoma sufficiently well to result in enucleation of the eye (case 8 and fig. 11).

That angioma can simulate malignant melanoma is obvious from the report of Hill and Dart.[13] A dramatic example is that given in case 9 in this paper (fig. 12). The tumor disappeared after enucleation because a considerable amount of blood had drained from the angioma. It seems reasonable to presume that digital pressure on the eye before enucleation will reduce the size or even obliterate the angioma and that this reduction can be observed by means of an ophthalmoscope. Unless future experience determines that this method is impracticable, it should

Fig. 11 (case 8).—*A*, area of localized melanosis. (This should be contrasted with *B*). *B*, normal area of choroid.

be a point in differential diagnosis, since an eye containing an angioma need not be enucleated.

Metastatic carcinoma of the choroid usually is diagnosed because of the history, yet several instances are on record in which this condition was mistaken for malignant melanoma. Correct diagnosis will save the patient the necessity of an enucleation, and by means of roentgen or radium therapy [14] useful vision may be retained for the remainder of the patient's life. Greenwood [15] has been able to diagnose carcinoma by its appearance alone. He has found that carcinoma appears as a rapidly growing gray or white flat mass rarely over 3 to 4 mm. high.

12. Doherty, W. B., Jr.: Am. J. Ophth. **10**:1, 1927.

13. Hill, E., and Dart, R. O.: Tr. Am. Ophth. Soc. **34**:122, 1936.

14. Lemoine, A. N., and McLeod, J.: Bilateral Metastatic Carcinoma of the Choroid: Successful Roentgen Treatment of One Eye, Arch. Ophth. **16**:804 (Nov.) 1936.

15. Greenwood, A.: Internat. Cong. Ophth. **1**:565, 1922.

Metastatic carcinoma was bilateral in 20.8 per cent of 153 cases.[14] The frequency with which the lung is involved (83 per cent) [14] makes a careful examination of the chest and a roentgenogram of the lungs advisable as an aid to differential diagnosis (cases 10 and 11 and fig. 13).

An apparent curiosity is the metastatic chorioepithelioma from a testicular tumor. Such a growth, taken to be a malignant melanoma

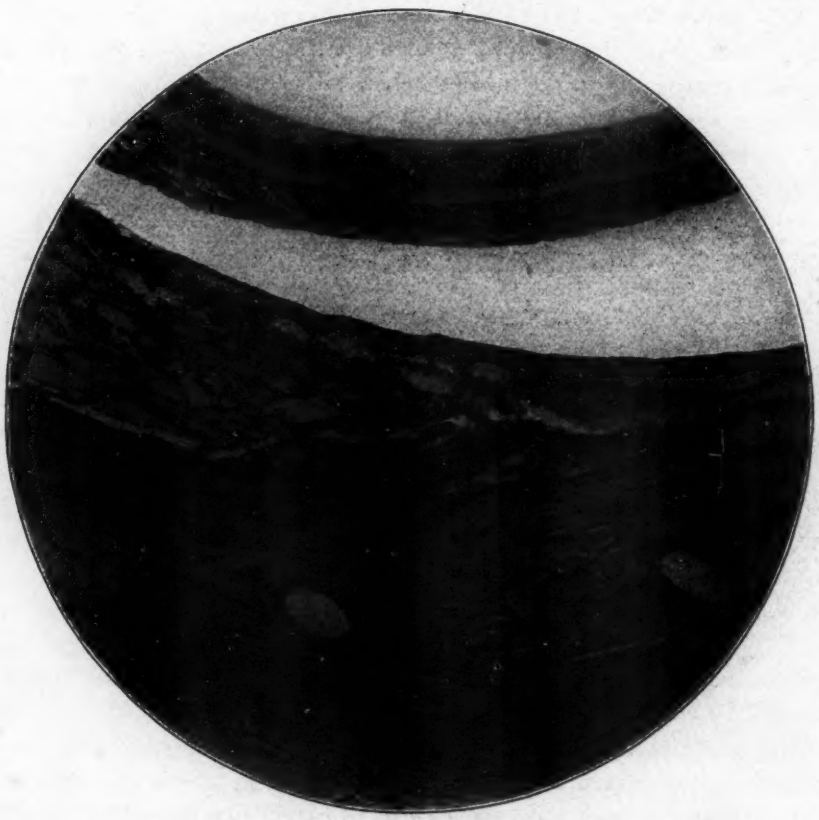

Fig. 12 (case 9).—Partially collapsed angioma of the choroid.

before enucleation, was reported by Reichling.[16] The same year MacDonald [17] made another report of metastasis to the eye from a chorioepithelioma of the testicle, but in this instance the clinical diagnosis was confirmed by a positive pregnancy test and later by necropsy. Such tumors contain syncytial cells. These publications stimulated a

16. Reichling, W.: Ocular Metastasis of a Chorionepithelioma, Arch. Ophth. **19**:156 (Jan.) 1938.

17. MacDonald, A. E.: Choroidal Chorionephithelioma Secondary to Teratoma of the Testicle, Arch. Ophth. **16**:672 (Oct.) 1936.

review of all specimens of malignant melanoma of the choroid containing giant cells and all specimens of carcinoma in the pathologic laboratory of the Massachusetts Eye and Ear Infirmary to see if by chance a chorioepithelioma had been diagnosed improperly. None was found. Recently a choroidal tumor developed in a 28 year old woman on whom an abortion had been performed a year previously. Clinical evidence of chorioepithelioma, including a pregnancy test, was negative. Examination of the enucleated specimen proved the tumor to be a typical malignant melanoma.

The fact that malignant melanoma can occur in the first decade of life suggests that retinoblastoma may be confused with a nonpigmented malignant melanoma. The retinoblastoma reported by Verhoeff [18] in the eye of a person 48 years of age is an example of this type of error. An error in diagnosis between retinoblastoma and malignant melanoma

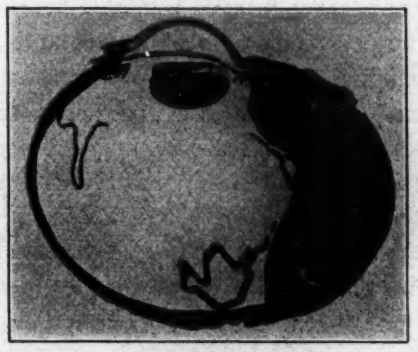

Fig. 13 (case 10).—Bilobate metastatic carcinoma of the choroid.

which results in enucleation at times may be of serious import, since it may mean removal of an only eye containing a small retinoblastoma that might have been held in check by roentgen or radium therapy for the remainder of the patient's life. Retinoblastoma does not produce uveitis, even when considerable necrosis is present.

Neurofibroma of the choroid, usually occurring in younger persons and often associated with neurofibroma of the orbit, exophthalmos and buphthalmos,[19] should be distinguished easily from malignant melanoma, yet Stough [20] reported a case of neurofibroma of the choroid in a patient 21 years of age in which the clinical picture was apparently that of a nonpigmented malignant melanoma. In a personal communication the

18. Verhoeff, F. H.: Retinoblastoma: Report of a Case in a Man Aged Forty-Eight, Arch. Ophth. **2**:643 (Dec.) 1929.

19. Wheeler, J. M.: Am. J. Ophth. **20**:368, 1937.

20. Stough, J. T.: Intra-Ocular Neurofibroma: Report of a Case, Arch. Ophth. **18**:540 (Oct.) 1937.

author stated that "the clinical characteristics would be no different from malignant melanoma." In another case reported by Freeman [21] in which difficulty in differential diagnosis might have arisen, a neurofibroma was found at autopsy in a man 56 years of age.

It is remotely possible that other conditions might be mistaken for malignant melanoma, the plausibility of which in some instances is strengthened by references to cases or case reports. These conditions are:

Benign melanoma of the choroid [22]
Meningioma
True glioma of the retina
Tuberous sclerosis
Angioblastomatosis retinae
Other even rarer retinal neoplasms

Benign melanoma, or nevus, of the choroid, is usually found by routine pathologic examination in eyes removed because of some other pathologic lesion. The possibility of benign melanoma, or nevus, of the choroid simulating malignant melanoma is ever present. These tumors, which may vary from ½ disk area to 4 disk areas, are sharply set off from the rest of the fundus and are homogeneously dark and of a roughly circular outline.[23] Cattaneo [24] has found that these benign tumors do not produce a scotoma. The growth in case 12 represents a beningn melanoma (figs. 14 and 15). The eye was enucleated because of a destructive lesion of the anterior segment, the melanoma being discovered in the course of the microscopic examination.

Cushing and Eisenhardt [25] reported a case of meningioma in a girl who had an intraocular neoplasm at 7 years of age, for which the eye was enucleated. A meningioma arising from the optic nerve sheath apparently had penetrated into the choroid, a most remarkable occurrence. Verhoeff made the diagnosis and referred the patient to Cushing for study and further treatment for intracranial involvement.

True glioma, or astrocytoma, of the retina has been observed and reported only once.[26] This tumor was present in a patient 23 years

21. Freeman, D.: Neurofibroma of the Choroid, Arch. Ophth. **11**:641 (April) 1934.

22. Benign melanoma more probably belongs under the list of conditions resembling malignant melanoma. It is placed here because no specific examples of benign melanoma simulating malignant melanoma are at hand.

23. Moore, R. F.: Roy. London Ophth. Hosp. Rep. (pt. 3) **19**:411, 1914.

24. Cattaneo, D.: Ann. di ottal. e clin. ocul. **54**:1097, 1926.

25. Cushing, H., and Eisenhardt, L.: Meningiomas, Springfield, Ill., Charles C. Thomas, Publisher, 1938, p. 287.

26. McLean, J. M.: Astrocytoma (True Glioma) of the Retina: Report of a Case, Arch. Ophth. **18**:255 (Aug.) 1937.

of age. The published picture does not resemble malignant melanoma. Although probably it would appear more like a retinoblastoma, it is conceivable that this condition might present a picture similar to a nonpigmented malignant melanoma which had invaded the retina.

In discussing the differential diagnosis in his case, McLean [26] mentioned primary retinal sarcoma but considered tuberous sclerosis as the only lesion at all similar to this tumor.

It is possible, but likewise improbable, that angioblastomatosis of the retina (Lindau's or von Hippel's disease) and retinitis with massive exudate could give some trouble in differential diagnosis. Their appearance, age of occurrence and behavior should tend to exclude them.

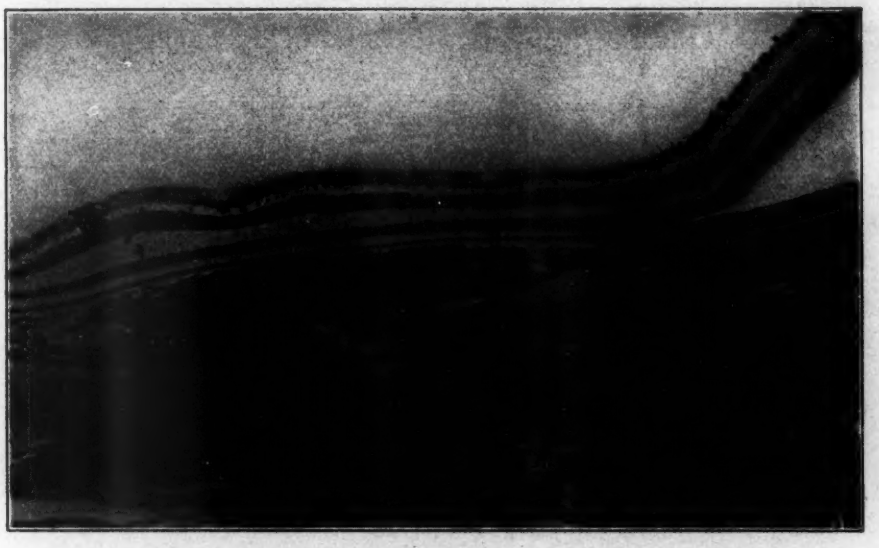

Fig. 14 (case 12).—Benign melanoma of the choroid.

EVALUATION OF DIAGNOSTIC SIGNS

If malignant melanoma is present in the eye, the only real protection to the life of the patient is enucleation, which in itself is not absolute, since occasionally even the smallest neoplasm may have already metastasized. It is important, therefore, to detect the neoplasm as early as possible and to enucleate the eye without delay, provided there is no evidence of metastasis already present. Doubtless instances do exist in which death from metastasis occurs without the discovery of the primary site of the tumor in the eye, since removal and examination of the eyes are almost never included in necropsy studies. Ewing [27] stated that one third of all malignant melanomas have their origin in the eye.

27. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 931.

Discovery of malignant melanoma should be made during routine examination of the eye before the tumor is large enough to produce signs and symptoms recognizable by the patient. The possible existence of malignant melanoma, therefore, must be borne in mind in every patient studied, but more particularly after the second or third decade of life. Having this constantly in mind, the examiner habitually makes thorough ophthalmoscopic examinations.

Ophthalmoscopic Findings.—The most important single diagnostic criterion is the ophthalmoscopic appearance of the tumor. Often the tumor is immediately subjacent to the retina at some place. To see that area ophthalmoscopically is to recognize its true nature almost invariably.[28] Because of the simple non-neoplastic proliferation of the pigment epithelium of the retina, stimulated by the neoplasm, the area

Fig. 15 (case 12).—Bleached section showing the benign character of the tumor cells.

usually looks darkened, even in association with leukosarcomas. Blood vessels in the choroid or at times in the neoplasm itself may be seen through the retina as evidence that solid tissue is immediately beneath the separated retina. The exact appearance in individual cases varies greatly. When the retina is not largely separated, the tumor appears to be a relatively flat or round mass. It cannot be reduced by pressure on the eye. To prove the absence of solid tissue behind the separated retina, the Friedenwald ophthalmoscope has been found useful. Relatively optically clear fluid behind a retinal separation is demonstrated by the presence of the illuminated streak on the choroid as well as on the retina. An "outstanding beam" may also be observed in the subretinal

28. Evaluation of the true importance of the appearance of the fundus and of the faults of transillumination in diagnosis has long been discussed by F. H. Verhoeff in his lectures. So far as I can determine, it has not been published.

fluid, depending on the opacity of the fluid.[29] The separated retina secondary to the malignant melanoma is neither tremulous nor arranged in hills and valleys. The location of the separation of the retina associated with malignant melanoma is not reducible by rest in bed, nor is its position shifted by gravity.

Opacities of the vitreous are rarely found in association with uninflamed choroidal neoplasms. Their presence indicates some other pathologic process.

Visual Fields.—The small malignant melanoma arises in the more external portions of the choroid, only later invading the choriocapillaris.[30] It is evident that a small neoplasm not yet invading the choriocapillaris would not alter the function of the retina sufficiently to produce a scotoma. At this stage benign and malignant melanomas could not be differentiated by study of the visual field.

Malignant melanoma may have its origin in or near the fovea, so that central vision is destroyed early. This alone may be productive of early diagnosis should the patient become conscious of diminished vision.

Malignant melanoma would tend to produce a demonstrable change in the visual field as soon as a sufficiently large amount of the choriocapillaris is replaced by tumor. A lesion of appreciable size, similar in appearance to a malignant melanoma, which does not give rise to a scotoma is most probably a benign melanoma, although instances of benign melanoma replacing the choriocapillaris have been seen. Separation of the retina, which is almost invariably present with the larger malignant melanomas, contributes greatly to the size of the scotoma.

Transillumination.—Transillumination, or diaphanoscopy, first used by Lange[31] in connection with the eye in 1881, offered a real advance in the diagnosis of malignant melanoma. At that time the source of illumination was a gas flame, the light being focused by means of oblique illumination. The discovery of the electric light brought great refinements in technic, although the early electric lamps produced so much heat that cooling by a continuous flow of water in the instrument itself was necessary. A curved glass tube carrying sufficient light by reflection to be of practical use permitted Stephens[31] to transilluminate the eye from behind the equator. Lancaster[32] designed an electric posterior

29. Friedenwald, J. S.: Clinical Studies in Slitlamp Ophthalmoscopy, Arch. Ophth. **1**:575 (May) 1929.

30. Ginsberg, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1928, vol. 11, pt. 1, p. 515.

31. Cited by Wood, C. A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 5, p. 3938.

32. Lancaster, W. B.: Tr. Am. Ophth. Soc. **13**:445, 1913.

transilluminator which could be placed in almost any desired position behind the globe through an incision in Tenon's capsule. Greenwood [33] stressed the value of this instrument. Burke [34] demonstrated how the details of the fundus could be seen by means of transillumination when a bright light was used against the sclera, provided the examiner's eye was close to the patient's eye and provided further that both the patient and the examiner were emmetropic.

The value of transillumination in the diagnosis of malignant melanoma is limited.[28] First, differentiation cannot be made by transillumination between hemorrhage, opaque exudate, angioma, benign melanoma, malignant melanoma and other pathologic processes of the eye giving rise to opaque tissue or exudate. All would appear dark. Second, unpigmented malignant melanomas may be transilluminated

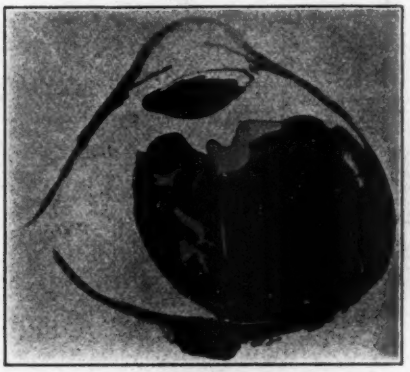

Fig. 16 (case 13).—Eye containing a large nonpigmented malignant melanoma of the choroid in which clear transillumination was obtained before and after enucleation. The heavy staining of the tissue, not melanin, is responsible for the dark color.

clearly (case 13 and fig. 16). Third, transillumination of the posterior segment of the globe necessitates hospitalization in order to place the transilluminator in Tenon's capsule.

In spite of these limitations, transillumination is of real value if it only gives confirmation to the diagnosis in most instances. Transillumination should make a real differentiation between senile and juvenile types of disciform degeneration of the macula, a matter of great importance, because the juvenile type, due to serous, not hemorrhagic, exudate, tends to heal (as has been brought out). When the interior of the eye cannot be seen, the presence of a tumor can be detected clinically only by altered transillumination.

33. Greenwood, A.: Tr. Am. Ophth. Soc. **13**:503, 1913.
34. Burke, J. W.: Tr. Am. Ophth. Soc., **34**:239. 1936.

There are three general methods of transillumination: (1) observation of a red reflex or general illumination of the pupillary area; (2) study of the details of the interior of the eye, as in ophthalmoscopy, by means of illumination through the sclera as suggested by Burke,[34] and (3) Weve's [35] method of the introduction of a focused beam of light through the pupil by means of indirect ophthalmoscopy while an assistant observes the sclera for the amount of transillumination.[36]

If the interior of the eye can be seen, careful observation of the lesion by means of Burke's[34] method of transillumination is to be preferred because it is more accurate and more delicate. Small variations in transillumination not detectable by the first method are easily observed with this method. Lack of emmetropia in the eye of the patient or of the observer is often remedied by the use of an ophthalmoscope, but of course the ophthalmoscope light is not used. The ophthalmoscope lens system further facilitates the study of superficial details in an elevated pathologic lesion. By use of this method, slight amounts of reduction in transillumination in leukosarcoma and nonpigmented lesions might be differentiated from separations of the retina. It was by means of this method that the correct clinical diagnosis was established in case 1.

Lack of experience precludes any discussion of the third method of transillumination.

A really satisfactory study of the eye by transillumination necessitates proper placement of the light. It must be placed on the sclera so that the opaque lesion is between the light and the observer or between the light and the part observed. Failure to place the light in exactly the proper position can be misleading, especially if the light is bright, since a considerable amount of diffusion of the light in the coats of the eye over a wide area may illuminate a large area brightly, giving a false impression of clear transillumination. Again the light can be too bright, giving rise to considerable diffusion, which will prevent observation of a small or slightly opaque lesion. This is especially true when the first

35. van Manen, J. G.: Die diathermische Behandlung der Netzhautablösung in der Universitätsaugenklinik Utrecht und ihre Ergebnisse im Jahre 1935, Utrecht, Keminken Zook, 1936.

36. This method was used by Weve and associates as a means of locating accurately on the sclera the correspondence of the hole in the retina for operation on the separated retina. It necessitates an opening into Tenon's capsule and the use of retractors. It may necessitate the cutting of an extraocular muscle if the suspicious area to be tested with transillumination is in the macular region or near the nerve head. Although I have used the method in the diagnosis of malignant melanoma once, with only moderate satisfaction, no doubt it could be developed with practice and refinements, such as use of a laryngeal type of mirror or with a properly developed endoscope to facilitate observation.

method of transillumination is used. Finally, it is possible to have too large a source of light, particularly if the lesion is small.[37]

An attempt has been made to design a satisfactory transilluminator for use in the first two methods of transillumination that is adaptable to use in the conjunctival sac or as a posterior transilluminator in Tenon's capsule.[38]

Appearance of the Iris.—Vascularization of the iris typical of hemorrhagic glaucoma is observed occasionally in instances of sarcoma with glaucoma.

Laboratory Tests.—A diagnostic test for malignant melanoma seemed possible on the basis of Zondek's and Krohn's [39] erythrophore test for intermedin, which is performed by injecting an acetone precipitate of

TABLE 2.—*Summary of Data on Differential Diagnosis*

Conditions Hiding Malignant Melanoma	Diagnostic Criteria
Glaucoma	Unilateral; not amenable to treatment
Separated retina	Smooth, nontremulous retina; viscous subretinal fluid
Uveitis	Unilateral; does not subside
Conditions Simulating Malignant Melanoma	
Disciform degeneration	Often bilateral; sharp edge; no increase in size
Metastatic carcinoma	Unpigmented tumor; rapidly growing and flat; often bilateral; associated with metastasis in lung
Metastatic chorioepithelioma	Positive pregnancy test; neoplasm of gonads; probably other general metastases
Angioma	Reducible by pressure on eye
Localized inflammation	Opacities of vitreous
Retinoblastoma	Often bilateral in very young; calcium often demonstrable by roentgen examination
Neurofibroma	Often present in young with buphthalmos
Separation of choroid	Clear transillumination; soft eye; deep anterior chamber
Localized melanosis	No increase in size; no scotoma
Benign melanoma	No increase in size; usually no scotoma
Subchoroidal hemorrhage	No increase in size
Glioma of retina	
Other rare tumors	

the blood into a certain type of fish. The fish (Elritzen) shows ventral red spots as a result of expansion of erythrophores, a change normally characteristic of the spawning season. Lund [40] investigated this problem thoroughly, giving it up because of failure to obtain any consistent results. Should an accumulation of abnormal materials reach the body fluids from the neoplasm in sufficient quantity to be measured it is probable that the tumor would have already metastasized, because the size of the eye would limit the size of the tumor.

37. These observations are based in part on the results of routine transillumination of several hundred enucleated eyes in the laboratory.

38. A description of the transilluminator designed by Mattis and myself will be published (Arch. Ophth.).

39. Zondek, B., and Krohn, H.: Klin. Wchnschr. **11**:405, 1932.

40. Lund, C. C.: Personal communications to the author.

Examination of the urine for melanin should be done. If the reaction is positive, it indicates most probably that a massive metastasis is already present.

Roentgen studies of the eye as yet have not given information of diagnostic value in malignant melanoma, though it is of diagnostic value in retinoblastoma.[41]

General Medical Examination.—General physical examination for evidences of metastasis is advisable as a diagnostic adjunct. It may be enhanced by a peritoneoscopy as advocated by Ruddock.[42]

REPORT OF CASES [43]

CASE 1.—A woman 43 years of age first consulted her ophthalmologist in February 1935 because of poor vision in the right eye. She sustained a contusion to the right eye when she ran into the corner of a bureau in September 1936. Some two weeks later she first discovered visual deterioration. Examination revealed a large separation of the retina on the temporal side. A diagnosis of simple separation was made.

On Feb. 23, 1937, a Šafář operation was performed, at which time no fluid was obtained after the Walker pins were withdrawn. No reduction in the separation resulted.

On March 3 a second Šafář operation gave no better results. Failure to obtain fluid or to soften the eye caused the surgeon to open the sclera widely by means of a large cautery puncture, but even then no fluid was obtained. After convalescence, the separation seemed unchanged. I saw the patient in consultation at this time, and in spite of only slight reduction in transillumination I made a diagnosis of malignant melanoma of the choroid based on the appearance of a solid mass with increased pigmentation immediately subjacent to the separated retina. The eye was enucleated on March 19.

After routine fixation,[44] the eye was opened. A relatively unpigmented pedunculated tumor was found on the temporal side with separation of the retina surrounding the neck of the tumor. Microscopic study of the tumor showed a type B spindle cell malignant melanoma of the choroid with invasion of the retina. Many of the operative wounds were identified. The older ones appeared as vascularized scar tissue somewhat similar to the normal sclera in staining reaction. The more recent wounds were filled with younger fibrous tissue. The tumor was not invading a single operative wound.

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41. Pfeiffer, R. L.: Tr. Am. Acad. Ophth. **40**:131, 1935.

42. Ruddock, J. C.: West. J. Surg. **42**:392, 1934.

43. Several of the ophthalmologists who supplied me with clinical material are purposely not mentioned. The errors in diagnosis that have arisen seem natural in view of the many hazards of diagnosis which this condition presents. No criticism is intended.

44. Routine fixation is done with a 10 per cent dilution of formaldehyde for forty-eight hours and hydrochloric acid (3 per cent) and alcohol (70 per cent) for sixteen hours. The eye is then opened, dehydrated in graded alcohols and embedded in pyroxylin. When the pyroxylin is sufficiently firm, sections are made and stained, usually with hematoxylin and eosin. This technic was used for all eyes except the eye in case 11, which remained in the solution of formaldehyde for a longer period.

CASE 2.—A woman 55 years of age came to the clinic of the Massachusetts Eye and Ear Infirmary in November 1937, complaining of flashes of light before the left eye of about two months' duration. Some four weeks after the first symptoms she sustained an injury over the left eye when she "banged" her head. One week after the injury the vision became poor in the left eye.

On examination it was found that the vision of the left eye was 20/100 and that there was a large ballooning separation of the retina in the upper temporal quadrant of the fundus and a somewhat flat separation over a large area of the retina, the total separation extending approximately from 11 to 9 o'clock. There was a corresponding defect in the visual field. No hole was found in the separated retina after prolonged and repeated studies. The right eye was normal.

Early in December 1937 a Šafář operation was done, at which time some 60 Walker points were inserted through the sclera from 3 to 9 o'clock at a distance from 8 to 16 mm. behind the limbus. It was observed that the operation did not reduce the amount of the separation. A recent communication from the surgeon stated: "Regarding the operation of Mrs. V., I can say that I do remember the case well because of the unsatisfactory result and my keen disappointment that she did not get along better than she did. I do distinctly remember at the operation that we tried hard to obtain fluid but were unable to do so."

No further treatment was offered the patient until September 1938, at which time the aqueous was turbid, the tension was elevated to 33 mm. (new Schiötz), posterior synechiae were present and transillumination was faulty in the upper temporal quadrant of the eye. A diagnosis of malignant melanoma was made, and the eye was enucleated.

After routine fixation, the eye was opened. A relatively large, rounded, heavily pigmented tumor of the choroid was present in the upper temporal quadrant. It was 17 mm. in diameter and projected into the vitreous some 10 mm. It was attached from near the ora serrata to well past the nerve head. Microscopic study proved the tumor to be a type B spindle cell malignant melanoma of the choroid containing little necrosis and slight inflammation. Many blood spaces were present. Operative wounds through the sclera were identified. Each of these old wounds was healed with vascularized scar tissue. Along only one wound was the tumor found to have extended. It had grown one third of the distance through the sclera. In some instances the vessels in the scar were few, in which event the healed scar resembled normal sclera closely. In fact, it is probable that some of the healed operative wounds resembled normal sclera so closely that they were not recognized. At the areas of puncture where the tumor had not replaced the choroid, healed scars of the choroid were evident as a part of the operative wounds.

An anterior peripheral synechia was present, obliterating the normal iridocorneal angle. Hemorrhage was found in the vitreous. The retina was separated over an extensive area on the side of the tumor by a heavy coagulum.

CASE 3.—A man 59 years of age came to the clinic of the Massachusetts Eye and Ear Infirmary in December 1937. He complained of awakening four days previously with a severe headache on the right side, associated with severe pain in the eye and swollen lids. Chemotic conjunctiva was protruding through the palpebral fissure. The cornea could be seen only with difficulty. It was hazy. The eye was blind. A diagnosis of panophthalmitis was made, and the patient was admitted to the hospital for removal of the eye. No source of infection from which a metastatic panophthalmitis could have originated was found, although it was discovered that the patient had mild diabetes. The surgeon, after considering the advisability of evisceration of the globe, finally performed enucleation instead.

After routine fixation, the eye was opened and a large somewhat pedunculated tumor of the choroid was found. Microscopic study showed that a large, rounded, highly necrotic mixed cell type (fascicular and epithelioid) of malignant melanoma of the choroid extended from the disk to the equator. The tumor was some 11 mm. in diameter. Considerable necrosis, inflammation and hemorrhage were present in the tumor. The neoplasm contained many blood spaces. It had penetrated into the retina through a hole in the lamina vitrea.

The anterior and posterior chambers were filled with an acidophilic coagulum infiltrated heavily with pus cells, moderately with free pigment and slightly with hemorrhage. There was a somewhat similar infiltration of the vitreous. The anterior choroid was also infiltrated with pus cells. Even the cornea contained an abnormally large number of wandering cells. The iris and ciliary body showed considerable hyperemia. The vessels of the limbus and bulbar conjunctiva were engorged markedly.

Glaucoma was evidenced by a broad anterior peripheral synechia, hyalinization of the ciliary processes, fibrosis of the iris and cupping of the optic nerve.

CASE 4.—A man 77 years of age came to the clinic of the Massachusetts Eye and Ear Infirmary in November 1937 with a large hypopyon ulcer of the left cornea. About 1 mm. of the cornea near the limbus was uninvolved by the ulcer. A small descemetocele was present somewhat below the center of the cornea. The hypopyon filled three fourths of the anterior chamber. Vision in the eye was reduced to bare perception of light. The patient presumed that the visual deterioration came on as a part of the inflammation of the eye some three weeks previously. There being no hope of useful vision with healing of the ulcer, the eye was removed in order to shorten the patient's convalescence.

After routine fixation, the eye was opened. A rounded, slightly pigmented neoplasm was found in the fundus surrounded by a separation of the retina. Microscopic study revealed the tumor to be a malignant melanoma of the choroid of the epithelioid type with extension into the optic nerve. The extensive hypopyon ulcer had no unusual or remarkable characteristics.

On discovery of the extension of the neoplasm deeply along the optic nerve, the patient was recalled with the intention of exenterating the remaining orbital tissue. In view of an enlargement of the liver, probably due to metastasis, and an area of increased density in the lungs, more like tuberculosis than a malignant growth, as well as the patient's age and the high nonprotein nitrogen content of the blood, no further operations were done.

CASE 5.[45]—A woman 24 years of age saw a black spot before the right eye in the center of vision in January 1934. This reduced her vision to 6/60. A large central scotoma was present. A somewhat rounded, slightly elevated, dark pigmented area about 1 disk diameter in size was present in the macular region. Peripheral to this was a rounded light area demonstrable only by indirect illumination, obtained by throwing the image of the incandescent filament of light to one side of the lesion. A diagnosis of malignant melanoma was made by the patient's ophthalmologist, and the eye was enucleated.

After routine fixation, the eye was opened. The macular lesion was like that seen with the ophthalmoscope except for the disappearance of the surrounding "edema" and the absence of red color in the background of the fundus.

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45. This case was referred to in my discussion on the paper of Verhoeff and Grossman (Arch. Ophth. **19**:468 [March] 1938).

Microscopic study revealed that the macular lesion was 0.9 mm. in diameter and 0.3 mm. high, located between the rods and cones and the lamina vitrea. Its inner surface was covered almost completely by an abnormal irregular pigment epithelium of the retina, some of the pigmented cells being piled up in two to three layers, but in two of the sections near the center of the lesion the pigment cells were absent over an area of 0.3 mm. In this region the rods and cones were also absent, but the remainder of the retina appeared normal. The outer surface adjacent to the choroid was bounded incompletely by the lamina vitrea, in one section the lamina being absent over a distance of 0.1 mm. An incomplete irregular row of newly proliferated pigment cells adjacent to the lamina vitrea extended some 0.4 mm. on one side and 0.2 mm. on the other side, but it did not extend along the central portion.

The main mass of the lesion consisted chiefly of young fibroblasts at various stages of development. Some of the cells were drawn out with long narrow processes attached; others were much more immature, the processes being short, and still others showed a character like that of connective tissue epithelioids, being almost round. Amorphous material, serum and melanotic pigment in round granules and small rod-shaped forms were scattered through the mass. Some of the pigment particles were in phagocytes. Although a few macrophages were present, the lesion itself seemed free from inflammatory cells in any appreciable number.

One part of the retina above the mass showed considerable edema of Henle's layer. A small subretinal hemorrhage was present at the periphery of the lesion, ranging from 0.3 to 0.5 mm. in width. The hemorrhage was flat, being not greater than 0.1 mm. thick. This subretinal hemorrhage with the resultant flat separation of the retina must have accounted for the larger area of abnormal appearance seen ophthalmoscopically by indirect illumination of the retina. The most likely source of this hemorrhage was from the choroid through the hole in the pigment epithelium, although the lesion itself contained little blood and no discernible blood pigment. At the base of the lesion where the lamina vitrea was absent a small blood vessel extended slightly into the lesion.

Under the lamina vitrea there was a dense infiltration of the choroid with many small lymphocytes and a few large lymphocytes. This area of infiltration was 0.5 mm. in diameter and 0.2 mm. thick. No vessels were seen in the choroidal inflammatory lesion. The choroid was not thickened appreciably in this area. No evidence of necrosis or organization was present in the choroid. At the periphery of the choroidal nodule of inflammation an emissarium extended through the sclera. This emissarium and its short posterior ciliary artery appeared to have no causative relation to the area of inflammation, since another similar nodule of chronic inflammation was present in the choroid near the nerve head unrelated to any emissaria or vessels.

The subretinal lesion represented a late stage of disciform degeneration of the macula. The healing process was rather advanced, and for that reason it is impossible to determine surely whether this lesion resulted from serum or hemorrhage under the pigment layer of the retina. Since this lesion was organized and since there was hemorrhage between the lesion and the retina, it probably belonged to the senile type.

CASE 6.—A woman 62 years of age had severe iridocyclitis in her blind left eye. The cause of the blindness was not obvious clinically. The aqueous contained many cells. The iris was markedly discolored and fixed. Although the interior of the eye could not be seen clearly, a hazy red reflex was visible everywhere except in the nasal portion, where the fundus appeared to contain a raised black mass

Transillumination was clear in all regions. The inflammation had been present for one month. I saw the patient in consultation and made a diagnosis of malignant melanoma in spite of clear transillumination anteriorly. The eye was removed some four days later.

After routine fixation, the eye was opened. An extensive separation of the choroid was present. The vitreous was largely fluid. Microscopic study showed that sclerosis of the iris meshwork was present although the angle was open. The anterior chamber was partially filled with an acidophilic coagulum containing many cells, some of which could be identified as polymorphonuclear leukocytes. The iris was moderately fibrosed and infiltrated with lymphocytes, plasma cells and a few macrophages. A moderate number of clump cells was present near the sphincter pupillae. Iris pigment on the anterior surface of the lens indicated that a synechia had been present but had later broken down. Some fibrinous exudate in the region of the pupillary margin was in the process of being organized into an incomplete pupillary membrane.

The lens capsule varied abnormally in thickness. Lamellar fibrillation of the anterior lens capsule was evident several millimeters peripheral to the pupillary area. The anterior capsular epithelial cells had extended back well past the equator.

The ciliary processes were heavily hyalinized. Nearer the bases of the processes a moderate amount of infiltration with lymphocytes and plasma cells was present. This infiltration extended into the circular fibers of the ciliary muscle.

An extensive separation of the ciliary body and choroid reached to the equator on the temporal side and to within 8 mm. of the nerve head on the nasal side. The uveal tissue above this separation was infiltrated with chronic inflammatory cells. Beneath the separation were a large number of macrophages heavily engorged with lipoids. Some small amount of subchoroidal exudate could be seen as an amorphous acidophilic material. A moderate amount of retinal separation was also present with the same type of exudate and phagocytes beneath it.

It is evident that the patient had an acute exacerbation of chronic uveitis with the formation of exudate beneath the choroid and the ciliary body. Exfoliation of the lens capsule and sclerosis of the meshwork of the iridocorneal angle were present. Glaucoma was not present probably because of the previous extensive hyalinization of the ciliary processes in part and because of the acute inflammation.

CASE 7.—In 1931 a woman 71 years of age insisted that cataract be removed from the left eye, which had been blinded many years previously by glaucoma. She desired a black pupil for cosmetic reasons. Her ophthalmologist advised enucleation, but he consented finally against his better judgment to remove the cataract. The cataract was removed in its capsule, but the ophthalmologist saw an elevated gray mass on the nasal side through the pupil by illumination of the ordinary hammer type, hand-operating light. This was considered to be a malignant melanoma. The patient was informed of this finding and was urged to have the eye removed. The eye was removed about twenty-one days later.

After routine fixation, the eye was opened. The vitreous was filled with blood. A large subchoroidal mass consistent with hemorrhage was seen. Microscopic changes typical of advanced glaucoma were present, consisting chiefly of a broad anterior peripheral synechia, cupping of the nerve head and loss of retinal ganglion cells.

Recent hemorrhage was present in the vitreous, but beneath the choroid on each side was a large organized hemorrhage with a considerable amount of recent hemor-

rhage infiltrating this tissue. The original cause of the subchoroidal hemorrhage was not obvious from the sections or from the history.

CASE 8.—A woman over 40 years of age had a relatively flat separation of the retina below. In this region no transillumination was obtained. The fundus could not be seen clearly because of opacities of the lens. A diagnosis of malignant melanoma was made by one ophthalmologist and confirmed by another. The eye was enucleated on Feb. 12, 1931.

After routine fixation, the eye was opened. A flat separation of the retina with fluid behind it was found below. There was no gross evidence of intraocular neoplasm. Microscopic study showed the eye to be highly pigmented; and in one region, below, the amount of choroidal pigment was much greater than elsewhere. This heavy pigmentation extended along an emissarium for a short distance. In bleached sections of this region the pigment-bearing cells appeared as normal chromatophores in unusually large numbers. The choroid was somewhat thickened in this region. A diagnosis of separation of the retina and localized melanosis of the choroid was made.

CASE 9.—A woman 42 years of age noticed a cloud before the right eye in January 1936. In March 1936 she went to an ophthalmologist, who found extensive separation of the retina. The visual field showed a corresponding defect. It was obvious to the ophthalmologist that a solid mass typical of malignant melanoma was behind the separated retina. After some hesitation the patient finally consented to have an enucleation done, and in March the eye was removed. After enucleation, the eye transilluminated clearly.

After routine fixation, the eye was opened. Grossly, there was no evidence of intraocular tumor or separation of the retina. The eye appeared to be normal. Microscopic study revealed a capillary angioma of the choroid some 3 mm. in diameter. The spaces of the angioma were largely collapsed. Obviously the angioma previous to enucleation gave the appearance of malignant melanoma.

CASE 10.—A man 49 years of age was observed by his ophthalmologist to have a large bilobate gray tumor from within 3 mm. of the nerve head to the margin of the fundus of the right eye. The tension was 48 mm. (new Schiötz). In June 1932 a diagnosis of leukosarcoma was made, and the eye was enucleated immediately.

After routine fixation, the eye was opened. From a microscopic study the diagnosis of metastatic carcinoma was made. Blockage of the filtration angle by an anterior peripheral synechia and slight cortical cataractous changes in the lens near the equator were present.

The patient was not followed by his ophthalmologist, and it has been impossible to obtain further information. The site of the original tumor therefore is not known.

CASE 11.—A 60 year old man who complained of impaired vision of six weeks' duration was seen in May 1928. After a diagnosis of tumor of the choroid of the left eye, enucleation was done.

The eye was opened, the presence of the tumor was verified grossly and a portion of the eye containing the tumor was sent to the pathologic laboratory of the Massachusetts Eye and Ear Infirmary in a 10 per cent diluted solution of formaldehyde to be sectioned. The tumor proved to be a large, somewhat flat, metastatic carcinoma of the choroid containing a large amount of necrosis.

A recent report from the attending ophthalmologist stated that the patient died in October 1928. At autopsy carcinoma was found to be present in both lungs and in the liver. The primary focus was considered to be in the liver. The patient presented no general clinical symptoms prior to the enucleation of the eye.

CASE 12.—A man 24 years of age sustained a severe lime burn of the conjunctiva and cornea in 1920. This resulted in a leukomatous cornea and an extensive symblepharon. In 1922 the eye was enucleated.

After routine fixation, the eye was opened. Microscopic study revealed a benign melanoma of the choroid just temporal to the fovea. It was 1 mm. long and 0.2 mm. high. It produced a thickening of the choroid at this point, involving the entire choroid except the choriocapillaris. The melanoma was pigmented heavily. Bleached sections showed large hexagonal-shaped cells rich in cytoplasm with small darkly staining inactive nuclei. There was no evidence of mitosis. No doubt this lesion could have been seen with an ophthalmoscope if the anterior segment of the eye had been transparent. Probably it would not have produced a scotoma, since the overlying choriocapillaris was uninvolved and the retina was normal in appearance.

A dense leukoma of the cornea was present. An anterior peripheral synechia and a moderate cupping indicated that glaucoma was present, although the glaucoma had not destroyed the retinal ganglion cells to any considerable extent.

CASE 13.—In March 1929 a separation of the retina extending downward and nasally was observed in the left eye of a woman 50 years of age. Although a tumor was suspected, transillumination was clear and unreduced. Because of the clear transillumination, the eye was not removed. However, some nine months later the eye became blind and painful. The tension was 100 mm. (old Schiötz). The eye was enucleated and brought to the laboratory by the ophthalmologist, who said he had a "hunch" a sarcoma was present, even in spite of the clear transillumination. After enucleation transillumination of the eye was clear.

After routine fixation, the eye was opened, and a relatively large "leukosarcoma" was found. Microscopic study revealed a B type spindle cell slightly pedunculated malignant melanoma of the choroid with considerable necrosis. Glaucoma was evidenced by an anterior peripheral synechia and glaucomatous cupping.

CONCLUSIONS

1. Failure to remove an eye containing a malignant melanoma is a greater mistake than removal of an eye erroneously thought to contain the neoplasm.

2. Malignant melanoma of the ciliary body and choroid are hidden frequently by a variety of disease conditions.

3. Malignant melanoma must be suspected in every instance of unilateral glaucoma, separation of the retina, iridocyclitis and endophthalmitis.

4. Hopelessly injured eyes and all painful blind eyes should be enucleated because they may contain malignant melanomas.

5. Operations to relieve pain and congestion of glaucoma in blind eyes are to be avoided; enucleation is the safer procedure.

6. Operation through the sclera for separation of the retina which does not obtain a flow of the subretinal fluid and does not reduce the amount of the separation suggests the presence of a choroidal neoplasm.

7. Evisceration of an eye possibly containing a malignant melanoma is to be avoided.

8. Malignant melanomas have simulated disciform degeneration of the macula, localized inflammatory tumors of the choroid, spontaneous separation of the choroid, subchoroidal hemorrhage, localized melanosis oculi, benign melanoma, angioma, metastatic carcinoma, metastatic chorioepithelioma, retinoblastoma and neurofibroma.

9. Differentiation of disciform degeneration of the macula from malignant melanoma is important because it does not necessarily destroy vision, it is often bilateral and it may tend to heal. This differentiation may be possible only by prolonged observation.

10. Angioma of the choroid may be observed to collapse partially from digital compression on the eyeball.

11. Metastatic carcinoma differentiated by flat unpigmented appearance, tending to be bilateral and to be present in the lungs, does not call for enucleation of the eye.

12. Metastatic chorioepithelioma can be differentiated by the presence of uterine, ovarian or testicular tumor and by a positive pregnancy test.

13. Retinoblastoma having been found in the fourth decade and malignant melanoma in the first decade of life, age is of no value in differentiation. One may have to rely on the ophthalmoscopic appearance and on the presence of calcium (psammoma bodies), as shown by roentgen examination, to differentiate between the retinal tumor and the nonpigmented malignant melanoma that has invaded the retina.

14. The most important single diagnostic criterion of malignant melanoma is the appearance of the fundus with evidence of a solid mass immediately beneath a nontremulous separated retina.

15. Mistakes in transillumination can result from improper placing of the light, too large a source and too bright a light.

16. Transillumination as a diagnostic method may be misleading; an unpigmented neoplasm (leukosarcoma) may be transilluminated clearly, many growths other than malignant melanoma may not be transilluminated.

17. Transillumination is the only available clinical evidence of the presence of an intraocular neoplasm when the interior of the eye cannot be seen.

18. More accurate information can be obtained by Burke's and Weve's methods of transillumination than by watching the pupil for general illumination of the interior of the globe.

19. Except when a malignant melanoma is extremely small, it gives rise to a defect in the visual field.

20. Vascularization of the surface of the iris characteristic of hemorrhagic glaucoma may also be associated with glaucoma secondary to malignant melanoma.

21. A general physical examination for evidence of metastasis and a test for melanuria should be a part of each diagnostic survey for malignant melanoma.

ABSTRACT OF DISCUSSION

DR. EVERETT C. MOULTON, Fort Smith, Ark.: Nomenclature as applied to malignant neoplasms of the choroid is at times confusing. One hears of aleukemic leukemia and now nonpigmented malignant melanoma. While I am thoroughly in accord with such terms, their usage seems to be about as involved as a double negative would be. I hope the pathologists will eventually clarify the terminology of malignancy in its ocular applications.

Dr. Terry's list of 94 cases of malignant melanoma of the choroid covers a period beginning forty years ago. This offers an explanation as to why the condition in exactly one third of them was erroneously diagnosed as glaucoma, and the tumor was not suspected clinically in these 31 cases. This percentage of error must have undoubtedly decreased in the more recent decades, for it has been learned that intraocular neoplasm should be suspected in every case of glaucoma, especially if only one eye is involved.

Probably all authorities will list malignant melanoma of the choroid as an absolute indication for the removal of the eyeball. Yet the responsibility of the oculist is great if that tumor is small and useful vision remains. In such an instance the diagnosis must be accurate. A few months ago a middle-aged woman presented such a condition. Surgical treatment for detachment of the retina had been advised. Vision at the time was 20/80. After a third consultant had confirmed the diagnosis of malignant melanoma, the eye was enucleated. The volume of the tumor was approximately 75 cu. mm., and the growth proved to be poorly pigmented. In such cases the intraocular tumor should be recognized ophthalmoscopically by its elevation into the vitreous, its sharp outline, the straight course of the retinal vessels over the surface of the tumor and usually a superficial pigmentation over the growth and a fresh retinal detachment at the border of the growth.

Some blind painful eyes, the result of chronic intraocular inflammation, when removed and opened are found to contain a malignant melanoma. Usually the tumor is necrotic, a condition which produces marked secondary inflammation of the iris and ciliary body. I have seen 1 such case in a 10 year old boy who had a suppurative exudate in the anterior chamber and opaque swelling of the iris accompanied by separation of the retina, with resulting blindness. Contrary to the case of Cohen, in which evisceration was done, this eye was enucleated; but local recurrence and death rapidly followed. This again opens the time-honored discussion of evisceration versus enucleation.

Dr. Terry mentioned retinoblastoma as a condition so closely resembling malignant melanoma that eyes have been enucleated because of a mistaken diagnosis. Ophthalmologists think of malignant melanoma of the choroid as a disease of adult life and old age and one that is so rare in childhood that in all probability every intraocular tumor in children should be considered a retinoblastoma. Recently I removed an eye of a 6 year old girl after a preoperative diagnosis of retino-

blastoma had been made. I was greatly surprised at the microscopic diagnosis of malignant melanoma. The tumor was scantily pigmented. The other eye was sound.

I wish to emphasize four important points made by the essayist, viz.: that malignant melanoma should be suspected in every case of separation of the retina, in every case of unilateral glaucoma, in every case of unilateral uveitis and in every case of a blind eye the fundus of which is not visible.

DR. ALLEN GREENWOOD, Boston: Dr. Terry has called attention to mistakes in the diagnosis of malignant melanoma and to methods for avoiding them.

In this day of frequent operation for the cure of separation of the retina the necessity of ruling out malignant melanoma before executing a diathermy operation is real and ever present. Before this time Professor Gonin's patients were not operated on as they are today, and therefore a malignant melanoma was more likely to be recognized as the condition progressed. If a separation of the retina is operated on and there is no escape of subretinal fluid or reduction of the separation, it is fairly certain that a tumor is the cause of the separation and that enucleation is necessary, as in the cases cited by Dr. Terry. With the improved ophthalmoscopes and the various types of transilluminators, added to a clear history, accurate differentiation should be made in the majority of cases.

A simple separation of the retina with fluid behind shows a movable retina with folds frequently present and a nearly normal color or a light gray. Tears or disinsertions are almost always present, and transillumination is the same in all positions of the transilluminator. If the separation is due to a tumor, the separation shows no tears or disinsertions, the highest point of separation is a darker color and is immobile and often the color of the melanoma shows through. The vessels are often unusually enlarged.

The differential diagnosis between malignant melanoma and metastatic carcinoma should not be difficult. Usually a history of a primary carcinoma elsewhere in the body is obtained, often of mammary carcinoma. Prostatic carcinoma, as in my first reported case, may be found only after death. All of the eight eyes that I have seen showed in the early stages of the choroidal involvement a flat, mottled, whitish tumor, with only a moderate elevation of the retina. There are often several of these tumors in the same eye, and they usually grow rapidly. I have likened the appearance of the flat carcinomatous growth to a flat piece of mottled, whitish marble lying beneath the retina. While the tumor grows rapidly, the separation of the retina outside of the growth is extremely slow. The tumor is also often bilateral, and this was so in 3 of my cases. Even a leukosarcoma, so-called, does not present the flat, white growth so characteristic of carcinoma. Dr. Terry spoke of roentgen examination of the lungs in making a differential diagnosis between malignant melanoma and carcinoma when there is an intraocular tumor. This is excellent advice, because in my experience the lungs are always involved prior to metastatic involvement of the brain and eye.

The only other thing which I wish to comment on is well covered by Dr. Terry and that is disciform degeneration in the macular region.

I have had no experience in doing an enucleation for this condition, suspecting it to be due to a malignant melanoma. The condition has been so distinctly a chorioretinal degeneration with hemorrhages and whitish areas that the diagnosis has been easily made. In such cases time will quickly demonstrate whether the growth is a malignant melanoma or not, though one does not wish to give too much time for a definite differentiation.

DR. CHARLES H. MAY, New York: I believe an equally interesting paper could be prepared on the subject of those eyes that have been enucleated for melanoma but in which no melanoma was found.

A differential diagnosis in some cases is difficult. Patients are sent from one consultant to another, and a certain number of consultants will say there is a tumor and an equal number will be doubtful. If one is dealing with a blind eye, it is easy to come to a conclusion. The patient has no use of the eye in any case, so why take a chance? Enucleation of the eye is the proper decision to arrive at in such an instance. But suppose one is dealing with an eye that has vision and there is a difference of opinion among competent ophthalmologists as to the nature of the changes in the back of the eye; this offers a difficult problem, and such instances are not at all rare.

I examined a woman of 40 who gave the history that she had noticed a reduction in vision during the previous month. Her vision was 20/50. The other eye was normal. I found a detached retina with sufficient indication of pigment behind the detachment to justify the diagnosis of melanosarcoma. My examination indicated the existence of a growth. There was a defect in the field corresponding to the situation of this detachment; transillumination was positive, and there was no increase in tension. Incidentally I wish to point out that it is commonly believed that there is always an increase of tension in cases of malignant melanoma of the choroid; this assumption is not corret; there is no increase in tension in the early stages, notwithstanding the statements found in various textbooks.

I have in mind a patient who presented sufficient signs to warrant a reasonably certain diagnosis; but the nature of the case indicated the advisability of consultation. Accordingly, the patient was referred to Dr. John E. Weeks, then practicing in New York, who reported "undoubtedly a growth." The patient was next referred to Dr. Robert G. Reese, who agreed with my diagnosis, and added "there is no question."

The eye was removed. There was no growth. A subchoroidal hemorrhage was found.

That brings up an ethical point: Should I have told the patient that there was no growth?

DR. THEODORE L. TERRY, Boston: I agree heartily with the objections made to the terminology, but I used that terminology deliberately. Medical terminology is one of the most unscientific tools. The terms I used, although not generally accepted, are to be preferred in the light of the present knowledge, or, had one better say, in the darkness of present ignorance.

An authentic case of astrocytoma or a glioma of the retina (a benign tumor) has been reported by McLean. Therefore, one cannot

use the term glioma of the retina in referring to a retinoblastoma. If I am sure that every one understands exactly to what I am referring, I usually speak of a primary malignant neoplasm of the uveal tract as a sarcoma. If there is any doubt, I much prefer to use the more specific, though less generally accepted, term, malignant melanoma. Until it is known from what tissue this tumor originates, one has no right to call it a sarcoma. It is not known that it is derived from mesoderm, and in calling it a sarcoma one is presuming that it is of mesodermal origin. It appears rather probable that the tumor may arise from the sheath of Schwann. In that instance one should say a schwannoma, or to make it a little more specific, a malignant schwannoma or a schwannoblastoma.

I thought as Dr. Moulton, that the percentage of failure to suspect the presence of the tumor would decrease, especially after the first paper on statistics by Johns and myself was published and the house officers at the Massachusetts Eye and Ear Infirmary became familiar with the problem. For a year or two practically every eye that came to the laboratory bore a diagnosis "question of malignant melanoma." However, that enthusiasm has passed, and one finds that the same mistake is occurring in approximately the same proportion.

It is never too early to remove the eye if a malignant melanoma is present. In some instances even the smallest malignant melanomas have given rise to metastases before the eye was enucleated. Enucleation is too late in some 30 to 40 per cent of the patients, since they die of metastases. For this reason failure to diagnose a tumor is a greater mistake than removal of an eye erroneously thought to contain a neoplasm.

In reply to Dr. May, the eye was removed in 13 cases because of a diagnosis of sarcoma, but no malignant melanoma was present. I should like to say again that an increase in ocular tension in this group was observed in 44 per cent. The ethical point that Dr. May suggests is a troublesome one to consider. It can be solved in some instances, when one knows that the condition present has destroyed the vision hopelessly, by telling the patient that it would be better to have the eye taken out, the necessity of a specific diagnosis thus being avoided.

Should an evisceration of the eye be done for any reason, the eviscerated materials should be subjected to a pathologic examination promptly.

CATARACT FOLLOWING THE INHALATION OF PARADICHLOROBENZENE VAPOR

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Two cases of opacification of the lens associated with jaundice and loss of weight occurring in young women following the inhalation of paradichlorobenze vapor suggested the toxic action of this drug on human beings and prompted some experimental investigations. The first patient, a 27 year old American housewife, was seen in the wards of the New York Hospital. She had been exposed to the fumes of this drug for two years. The second patient, a young housewife of 25 years, was seen in private practice. She had been exposed to the fumes of paradichlorobenzene for over a year.

Paradichlorobenzene, a by-product of the manufacture of trinitrophenol, is a white crystalline compound; it is slightly soluble in water and is soluble in alcohol, ether and benzene and closely resembles naphthalene in physical properties. The compound is chemically indifferent. The vapor is noninflammable, does not cling to fabrics, disappears rapidly on exposure to air and has a characteristic and not unpleasant odor.

Paradichlorobenzene is used extensively under many fanciful trade names as an agent to protect furs and clothing against moths. It is marketed in various types of paper containers as well as in "moth-bags," i. e., large bags impregnated with the "moth-repelling" chemical. It is sold in small tin containers designed to hang in closets. Some manufacturers make special containers to attach to vacuum cleaners in order to protect hangings and draperies. It is widely used both in commercial storage houses and in homes. It is easily obtainable over the counter of any drug or hardware store or grocery selling household accessories. The packages or containers in which it is sold at present show no statement concerning possible toxic qualities. The compound has also had some use in agriculture in protecting plants against insect pests. It is used to some extent as a deodorizing agent, for its not unpleasant odor can mask other odors.

There are few references in the literature to paradichlorobenzene, and most of these are merely passing remarks in the discussion of halogenated hydrocarbons.

Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 18, 1939.

Hitherto it has been mistakenly believed that the fumes of this drug were not poisonous to human beings and animals. Sollmann [1] stated that the compound is a powerful insecticide and is toxic to worms but that oral administration to dogs produces no toxic effects, even in doses of 1 Gm. per kilogram of body weight. Flury and Zernik [2] stated that the compound is practically without danger for man. In "Merck's Index" is the statement that the vapor is harmless to human beings and animals. Hamilton [3] stated that the addition of chlorine to the benzene ring changes the compound but little and that the monochlorinated compound is less toxic than benzene. No information is given in this source about the dichlorobenzenes. Heffter [4] stated that after the meta-dichlorobenzenes and the paradichlorobenzenes are fed to animals they are oxidized to the corresponding phenols and are withdrawn as paired sulfuric acid derivatives. The first indication of the possibility of the danger involved appeared in the "Encyclopedia of Industrial Hygiene" [5] in which there is a report of a case of fatal poisoning which occurred in Germany in a chemical cleaning works. Another report describes a case of severe poisoning in a person who lived on a floor above a fur cleaning establishment where large amounts of paradichlorobenzene were used.

REPORT OF CASES

CASE 1.—A 27 year old American born housewife came to the outpatient department of the New York Hospital on Nov. 6, 1936. She stated that for the previous five months she had suffered from weakness, dizziness and undue perspiration and had lost considerable weight. She noticed that for a few days prior to admission she had been jaundiced, had suffered several attacks of substernal oppression and had awakened at night gasping for breath.

She gave no history of recent infection or gastrointestinal upset and had noticed no change in the appearance of the urine or stools. She had been married five years and had had one abortion and no children.

Physical examination gave negative results, except for a definite jaundice and a a slight tremor of the hands. The basal metabolic rate on November 13 was + 6, and the reaction to the direct van den Bergh test was negative and to the indirect test slightly positive, with the icteric index at 18. The blood sugar on November 25 was 100 mg. per hundred cubic centimeters.

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1. Sollmann, T. H.: Manual of Pharmacology, ed. 5, Philadelphia, W. B. Saunders Company, 1936, p. 626.

2. Flury, F., and Zernik, F.: Schädliche Gase, Dämpfe, Nebel, Rauch- und Staubarten, Berlin, Julius Springer, 1931, p. 540.

3. Hamilton, A.: Industrial Poisons in the United States, New York, The Macmillan Company, 1925, chap. 3.

4. Heffter, A.: Handbuch der experimentellen Pharmakologie, Berlin, Julius Springer, 1923, vol. 1, p. 890.

5. Benzene Derivatives, in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1930, vol. 1, p. 238.

Over the course of the next two weeks the jaundice greatly deepened, so that hospitalization was advised, and the patient was admitted to the medical service of the New York Hospital on December 7.

In the hospital it was discovered that since September 1935 she had been using large amounts of paradichlorobenzene as a deodorant to counteract the odor of sewer gas arising from poor plumbing. The material was used on furniture, bedding, clothes, in the vacuum cleaner, over the door lintels and in various other places. She kept the windows of her house tightly closed but slept with her bedroom window open.

Physical examination revealed a well developed woman who was not in any apparent distress. The scleras were icteric, and the skin had a definite yellow tint. The edge of the liver was smooth and palpable 3 cm. below the costal margin. There was slight tenderness in the right upper quadrant at a point coinciding with the edge of the liver. The blood pressure was normal.

Examination of the eyes showed vision of 20/20 in each eye. The media were clear and the fundi normal. Slit lamp examination showed no lenticular changes. The visual fields were normal. The tension in each eye was 18.

The urine showed a faint trace of albumin, with occasional white and red blood cells (not a catheterized specimen). The bile test was negative. Millon's reagent gave a reddish coloration. A distinct pink coloration appeared after two to three hours' standing. This was due to phenol-like constituents in the urine.

The Wassermann test was negative. The blood count was as follows: 78 per cent hemoglobin (Sahli), 5,000,000 red cells and 12,500 white cells, with 46 per cent polymorphonuclear leukocytes, 30 per cent immature polymorphonuclear cells, 18 per cent lymphocytes and 6 per cent monocytes.

The fragility test of the blood gave normal results. The clotting time was twenty-five minutes; the bleeding time, 1 minute, and the clot retraction, fair with a moderately firm clot.

Chemical examination of the blood on December 7 showed 16 mg. of urea nitrogen per hundred cubic centimeters of blood and an icteric index of 10. On December 10 the icteric index was 21. On December 11 the uric acid content was 2.8 mg. per hundred cubic centimeters and the cholesterol content 302 mg.

The stool showed a positive reaction for urobilinogen. On roentgen examination, the Graham test showed no sign of disease of the gallbladder.

The patient improved while in the hospital and was discharged on December 24. She was followed in the outpatient department and continued in good health after discharge from the hospital, with, however, a recurrence of jaundice without any associated symptoms for a few weeks in March. She had used no paradichlorobenzene in her home since her discharge.

On Sept. 24, 1937, about a year and two months after the onset of her symptoms and one year after her removal from the fumes of the drug, the patient was referred to the ophthalmic clinic because of a sudden blurring of vision. She stated that during the previous week she had noted a rapid and increasing diminution of vision, especially in the right eye.

Vision in the right eye was reduced to perception of fingers at 3 feet (91 cm.). The vision in the left eye was 20/70, with no improvement with correction. The cornea of the right eye was clear, and the anterior chamber was shallow. Slit lamp examination showed a markedly swollen lenticular cortex with large water slits. The tension in the right eye was 30.

The cornea of the left eye was clear and the anterior chamber shallow. The cortex of the lens showed large dilated sutures with beginning opacifications in

the periphery. The fundus in this eye was normal. Extraocular movements were normal. The tension was 28. Within two weeks the lens became completely cataractous.

I operated on both eyes for removal of cataracts. Linear extractions were done, with visual acuity of 20/20 resulting in each eye.

CASE 2.—A young woman, aged 25, whom I saw three months before the writing of this report, stated that six months previously she suddenly became jaundiced and gradually lost about 14 pounds (6.4 Kg.) in weight. Her family doctor made a diagnosis of catarrhal jaundice. The jaundice cleared up after several weeks, and she regained some of her weight.

About a month before examination she noticed some blurring of vision in the left eye. On being questioned carefully, she stated that previous to the attack of jaundice it was her custom for over a year to do a great deal of sewing in a large closet which had no window. I discovered that there were always two cans of paradichlorobenzene in this closet for protection against moths. Several weeks before the jaundice appeared she recalled having had bronchitis, which I attributed to irritation from inhalation of the fumes of paradichlorobenzene. Since her illness, she has not been exposed to the fumes (a period of about six months). I was unable to obtain a satisfactory report of her medical condition at the time of the jaundice.

Examination of the right eye showed that it was entirely normal. The vision was 20/15, and the media, fundi and tension were normal. The vision of the left eye was reduced to 20/50. There were definite anterior cortical changes, especially in the periphery of the lens. The sutures were dilated. There was separation of the lamellae and several large water slits. There were no keratitic deposits or changes in the iris. The fundus was normal. Considering this finding unusual in a young person and recalling the first case reported here, I sent her to the New York Hospital, where, excepting for the changes in the lens, nothing unusual was found. There was no evidence of enlargement of the liver or phenol substance in the urine. The blood picture was normal. The opinion was that this patient showed the effects of a milder intoxication from the fumes of paradichlorobenzene than my first patient. She was advised as to general hygiene, placed on a diet high in vitamins and cautioned against exposing herself to the fumes of this agent. She was also told to make monthly reports.

At the last examination there was no change noted in her general condition excepting that she had regained her average weight. The right eye was still normal and the left unchanged.

EXPERIMENTAL RESULTS

An attempt was made to evaluate the toxicity of paradichlorobenzene. Since the uses of the compound are such that possible toxic effects would be due largely to the inhaled vapors, the compound was administered by inhalation to guinea pigs and rabbits. Facilities were lacking for quantitative determinations; consequently it could not be determined whether the animals received comparable amounts of paradichlorobenzene in the inhaled air or comparable amounts of ventilation in the cages. All observations made are solely of qualitative value.

Seven guinea pigs were used in the course of the experiment.[6] Animals 3, 4, 5 and 6 were placed in metal cages 30 by 30 by 45 cm., in which the open side was closed with a pane of glass. The cages were sealed as tightly as possible around the base, but no attempt was made to make them absolutely airtight. Several small holes were left for ventilation in the tops of the cages. The animals were kept in separate cages, all conforming to the same general type, and fed the same diets. In the cage in which animal 6 was kept, a small opening near the bottom of the cage, to which the animal had ready access, probably was a factor in its long survival. The animal died several days after the opening was plugged.

Animals 7, 8 and 9 were placed in cages formed from round cracker tins. The sheet of glass placed over each was raised ⅛ inch (0.31 cm.) from the edge of the tin to allow for ventilation.

Animals 3 and 9 were used as controls and were killed after a period longer than the longest time required for death in an exposed animal. They were kept in the same type of cage as was used for the other animals.

The paradichlorobenzene was placed in small gauze packages, 15 Gm. to each. Two or three packages were suspended from the top of each cage, out of reach of the animal. In cases in which the entire supply of the compound was exhausted before the animal died, a new supply was introduced into the cage. In the square cages 5 Gm. of the compound would vaporize in approximately two days. In the round cages in which ventilation was more restricted, the rate of vaporization was slower.

Most of the animals were killed when it appeared that death was imminent. Before death they showed a uniform picture of marked weakness. They were unable to stand or move around but made violent running motions when startled. The muscles showed coarse and fine tremors. Breathing was rapid and shallow. Guinea pig 7 died in the cage, and autopsy was performed several hours after death.

There was no gross pathologic change.

Microscopic examination showed pneumonic consolidation of a portion of the lungs in animal 5. Increased deposits of pigment were observed in the spleens of animals 4 and 5.

The most marked changes were in the liver. All the exposed guinea pigs showed vacuolation of the liver cells, involving all zones of the liver lobule but particularly the midzone of the lobule. There was a slight amount of damage to the liver cell in all cases, with shrunken,

6. The experiments on the guinea pigs were carried out by Dr. Wilbur G. Downs, in the Department of Public Health, Cornell University Medical College, under the supervision of Dr. Morton C. Kahn.

pyknotic nuclei in some of the cells. In guinea pigs 5 and 8 the damage was extensive, involving mainly the central portion of the liver lobule.

I exposed 4 rabbits to the vapor of paradichlorobenzene. The animals were placed in specially constructed boxes. After a month rabbits 1 and 2 began to show marked loss of weight, weakness and shallow breathing; they were killed when death was imminent. Rabbits 3 and 4, which were in a weakened condition, were removed from the boxes and returned to their cages. After a month they were again returned to the boxes for reexposure to lesser amounts of the fumes of the drug. This procedure was repeated three times over a period of six months, when rabbit 3 died suddenly. No lenticular opacities had appeared in any of the rabbits. No variation from the normal diet was made in any of these animals.

Rabbit 4 was removed from the box and returned to its cage. After two months the animal seemed to have regained considerable strength

Results of Experiments with Animals

Animal No.	Duration of Life After Exposure, Days	Gm. of Para-dichloro-benzene Used	Weight of Animal Before Exposure, Gm.	Weight of Animal at Death, Gm.	Difference Between the Two Weights, Gm.
4	8	40	593	...	...
5	11	50	651	446	205
6	47	205	541	419	122
7	19	30	530	372	158
8	5	13	585	448	137
Controls					
3 (not exposed)	50	None	617	714	97
9	65	None	440	445	5

and weight. It was then kept away from the influence of the fumes but was fed 5 Gm. of paradichlorobenzene daily by mouth, the drug being mixed with the food. Anorexia, loss of weight and general weakness soon reappeared. After three weeks I noted opacities of the lens in each eye. The cortex of the lens of the left eye began to swell rapidly. The animal grew perceptibly weaker and died during the fourth week. At postmortem examination the chief change found in these animals occurred in the liver. There were vacuolation of the liver cells and marked necrosis in the center of the lobule. No pathologic process could be observed in kidneys, lungs, adrenals or bone marrow.

Summary.—Paradichlorobenzene, inhaled in sufficient concentration, caused the death of guinea pigs and rabbits and produced a picture of vacuolation and necrosis of liver cells, principally in the central portion of the liver lobule.

No opacities of the lens appeared in any of these animals save in 1 rabbit which was fed the drug orally.

COMMENT

The great number of cases of cataract following the ingestion of dinitrophenol for the purpose of reduction of body weight aroused great interest in the entire subject of cataract produced by the toxic effect of drugs of the phenol-benzene groups. It was hoped that the study of the toxic effects in animals might give leads to the understanding of the mechanism of cataract formation in general. Naphthalene was one of the drugs first used to produce cataract in experimental animals. García Miranda [7] expressed the belief that the lesions in the lens were not due to metabolic disturbances and that naphthalene cataract always follows retinochoroidal lesions and must be considered as a complicated cataract. Bourne and Young [8] stated that naphthalene is detoxicated by the rabbit, partly by the conjugation with cysteine, and hence that the cataract which naphthalene produces may result from depriving the lens of cysteine, thus interfering with the metabolism of the lens.

In an earlier paper, Bourne and Campbell [9] found that naphthalene is unable to produce cataract in rabbits when the blood calcium is maintained at a high level, while with animals on a diet poor in calcium naphthalene produced extensive ocular lesion, including complete cataract. Kirby,[10] in his studies on the effect of alterations in the calcium content on epithelial cultures, stated that slight reductions were toxic but that increases within physiologic limits were not. Noting the fact that clinically in cataracts following dinitrophenol therapy the changes in the lens were subcapsular and that there was a rapid swelling of the cortex, Tainter and Borley [11] studied the possibility of changes in the permeability of the lens capsule. They found that dinitrophenol caused no increase in permeability of the capsules of beef lenses in vitro and of rat lenses in vivo. In most instances the appearance of cataract in patients in no way bore any relation to the amount of dinitrophenol taken, and they also observed that the changes in the lens occurred as a late manifestation, frequently appearing several months after the use of the drug was discontinued. There was also no relation in these cases between the appearance of the cataract and loss of weight. Dinitrophenol administered alone to experimental animals during their entire

7. García Miranda, A.: Contribución al estudio de las lesiones oculares naftalínicas, Arch. de oftal. hispano-am. **34**:76, 1934.

8. Bourne, M. C., and Young, L.: The Metabolism of Naphthalene in Rabbits, Biochem. J. **28**:803, 1934.

9. Bourne, M. C., and Campbell, D.: The Role of Calcium in Naphthalene Cataract, Brit. J. Ophth. **17**:220, 1933.

10. Kirby, D. B.: Pathogenesis of Senile Cataract, Arch. Ophth. **8**:97 (July) 1932.

11. Borley, W. E., and Tainter, M. L.: Effects of Dinitrophenol on the Permeability of the Capsule of the Lens, Arch. Ophth. **18**:908 (Dec.) 1937.

life span produced no lenticular changes. The question arose as to whether or not a vitamin deficiency predisposed these patients to the formation of cataract. Day, Langston and O'Brien [12] noted that a deprivation of vitamin B_2, or G, was consistently associated with the appearance of cataracts in rats. This observation was confirmed by Yudkin. However, Day and his co-workers [13] found that cataract did not occur in rats fed on a diet deficient in vitamin B_2, or G, provided that from 60 to 90 micrograms of pure riboflavin was added to the weekly diet. The exact action of riboflavin, an enzyme, is not understood.

It is a well known fact that the normal lens contains a relatively high percentage of vitamin C and that in cataractous lenses there is a decidedly lower concentration of this vitamin. The significance of this change is also not clear at present. Hawley and Pearson [14] concluded:

". . . in spite of the contrary evidence of other investigators, one is forced to conclude from the data here presented that the low content of vitamin C in the lens may be a result of cataractous changes rather than the underlying cause and that tissue saturation as measured by vitamin C content of the urine and blood suggests that it is probably not responsible for the onset of the cataract."

In order to investigate the problem of whether vitamin deficiency combined with the administration of dinitrophenol would produce cataract in experimental animals, Tainter and Borley [15] recently conducted a series of experiments with white rats and guinea pigs. They also used dinitrochlorbenzene, a chemical impurity in dinitrophenol. In all these experiments the animals that were suffering from severe or fatal deficiency of vitamin A, B_2 or C and were given dinitrophenol and dinitrochlorbenzene showed no demonstrable lenticular changes. Hence there was a widely uniform failure to produce by experimental means in animals cataracts similar to those reported as due to medication with dinitrophenol in human beings.

The question naturally arises as to whether or not the disturbed liver could play a role either directly or indirectly in the formation of cataract.

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12. Day, P. L.; Langston, W. C., and O'Brien, C. S.: Cataract and Other Ocular Changes in Vitamin G Deficiency: Experimental Study on Albino Rats, Am. J. Ophth. **14**:1005, 1931.

13. Day, P. L.; Darby, W. J., and Langston, W. C.: Identity of Flavin with Cataract-Preventive Factor, J. Nutrition **13**:389, 1937.

14. Hawley, E., and Pearson, O.: Vitamin C and Its Relation to Cataract, Arch. Ophth. **19**:959 (June) 1938.

15. Tainter, M. L., and Borley, W. E.: Influence of Vitamins and Dinitrophenol on the Production of Experimental Cataract, Arch. Ophth. **20**:30 (July) 1938.

RELATION OF HEPATIC DAMAGE TO CATARACT FORMATION

Hepatic damage may give rise to the following disturbances, which may play a role in the formation of cataract:

1. Liver is essential for the metabolism of the leucodehydrogenase or substances containing the SH group (betacrystallin). These compounds are necessary for processes of oxidation and reduction which go on in the lens (Ahlgren and Lewis[16]).

2. Liver is essential for the glutathione content of the lens, which is said to permit the lens to utilize oxygen (Krause,[17] Goldschmidt and Jess).

3. Liver is essential for vitamin C metabolism, which is said to be responsible for 30 per cent of the total reduction reactions of the lens as determined by the iodometric method. Sherman expressed the belief that vitamin C acts as a transport between organic metabolites and molecular oxygen (Müller;[18] Monyukova and Fradkin).

4. Damage to the liver may be responsible for changes in the permeability of the lens capsule by changing the p_H of the aqueous humor, the change being due to the marked acidosis accompanying severe damage to the liver.

5. Liver is essential for the formation of riboflavin (vitamin G, or B_2), which plays a role in the formation of cataract (its presence preventing formation), as shown by Day and co-workers, Yudkin, Sen and others.

6. Hepatic damage may disturb the formation of lens protein, since in severe damage protein metabolism is impaired. The two amino acids chiefly concerned in this respect are tryptophane and cysteine (Curtis, Hague and Kraybill[19]).

7. The change in p_H which occurs in acidosis subsequent to severe injury to the liver may affect the ionizable partition of the blood calcium, thereby predisposing to an increase in available calcium which may be taken up by lens (Makay and co-workers) or which may be responsible for the precipitation of protein material within the lens (Burga).

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16. Ahlgren, G.: Gibt es einen Stoffwechsel in der Kristallinse? Skandinav. Arch. f. Physiol. **44**:196, 1923. Lewis, H. P.: Chemistry of the Metabolism of the Compounds of Sulphur, in Luck, J. M.: Annual Review of Biochemistry, Stanford University, Calif., Stanford University Press, 1935, vol. 4, p. 149.

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18. Müller, H. K.: Reducing Property of the Aqueous Humor, Nature, London **132**:280, 1933; Kammerwasser und Linsenstoffwechsel, Arch. f. Augenh. **108**:41, 1934. Monyukova, N. K., and Fradkin, M. Y.: New Experimental Data on the Pathogenesis of Cataract, Sovet. vestnik oftal. **5**:97, 1934.

19. Curtis, P. M.; Hauge, S. M., and Kraybill, H. R.: The Nutritive Value of Certain Animal Protein Concentrates, J. Nutrition **5**:503, 1932.

8. The role of water balance may be affected by hepatic damage (as yet not clearly understood), which may induce shrinkage or swelling of the lens due to osmotic pressure variants (similar to the mechanism occurring in diabetes).

9. Toxic substances from the degenerating liver parenchyma may act either directly on the lens or on the parathyroid gland, thus indirectly producing an upset in the physiologic processes which normally take place in the lens.

10. Toxic poisons, such as arsenic and phosphorus, which provoke edema by injury to the capillaries, also affect the parenchyma and capillaries of the liver. Serious disease of the liver is associated with interference of water metabolism, and edema may result. The liver may have a diuretic controlling hormone corresponding to the antidiuretic hormones of the posterior lobe of the pituitary body. The modus operandi of these hormones is still not understood. Dr. Pick stated:

> The want for water by the blood and tissues, the dehydration of blood and tissue edema are controlled not only by osmotic and physico-chemical forces that regulate swelling and dehydration but also by a more rapid functioning control process which by the way of the vegetative mid-brain controls water metabolism either nervously or humorally through the various endocrine bodies.

11. A combination of the foregoing possibilities may lead to cataract formation.

SUMMARY

Two cases of jaundice, loss of weight and cataract formation occurring in young women are presented. The history in each case suggests a toxic hepatitis due to continued exposure to the fumes of paradichlorobenzene. The changes in the lenses appeared as a delayed reaction several months after the patients were removed from the influence of the fumes of this drug, a phenomenon strikingly similar to that noted in cataracts due to the toxic effects of dinitrophenol. Paradichlorobenzene inhaled in sufficient concentration caused the death of guinea pigs and rabbits and produced a picture of vacuolation and necrosis of liver cells, principally in the central portion of the liver lobule. No lenticular changes appeared excepting in 1 experimental rabbit which was fed the drug orally.

CONCLUSIONS

1. Paradichlorobenzene inhaled in sufficient quantities may produce severe toxic effects in human beings and in animals.

2. In human beings these toxic effects are manifested as a hepatitis, loss of weight and later cataract formation.

3. A warning of the toxic properties of this drug should be made, first, in order that manufacturers and distributors of this product shall be required to label containers properly that are sold to the general

public and, secondly, in order that the medical profession and health authorities shall be apprised of the danger of prolonged inhalation of the fumes of this substance.

ABSTRACT OF DISCUSSION

DR. JAMES E. LEBENSOHN, Chicago: Besides the cases cited by Dr. Berliner, many additional reports have demonstrated the potential toxicity of paradichlorobenzene. In "Queries and Minor Notes" in *The Journal of the American Medical Association,* the following interesting cases have been discussed ([*a*] **104:** 1028 [March 23] 1935; [*b*] **107:** 607 [Aug. 22] 1936).

(*a*) In March 1935, Dr. T. A. Claytor, of Washington, D. C., was called to examine a previously healthly woman in the twenties who had spent the entire day before packing winter clothing with crystals of paradichlorobenzene. Her bedroom adjoined the clothes closet, the door of which did not close tightly. In the morning she had gained 6 pounds (2.7 Kg.) due to swelling of the hands, feet and ankles, but after three days she returned to normal weight. Dr. Claytor recently informed me that the patient remained completely well and that no ocular disturbances developed.

(*b*) In August 1936, Dr. J. P. Nill, of McKeesport, Pa., wrote of a woman overcome in her home after a solution of this chemical had been sprayed under pressure into the wood to destroy termites. When he was consulted two days later, the patient complained of nausea, dyspnea, sleepiness, weakness and pain between the scapulas. The carbon dioxide–combining power of the blood was markedly reduced, and hematuria was noted for two days. At my request, Dr. Nill investigated the present condition of the patient and reported that recovery was permanent and that no ocular disturbance ever resulted.

To Dr. Berliner belongs the credit of being the first and only observer to discover cataracts produced by this agent. It is noteworthy that his patients were young, ages 25 and 27, and were exposed to the fumes of paradichlorobenzene for one and two years, respectively. In a personal communication, Dr. Howard C. Knapp, of East St. Louis, Ill., mentioned that cataract had developed bilaterally in a man of 37 who worked occasionally with dinitrochlorobenzene; however, the cause and effect relation in this case is questionable, for this is the only instance in which cataract has been attributed to this chemical, though the companies in East St. Louis have been handling large quantities of it for some years.

Dr. Berliner's experimental work confirms a recent study of G. R. Cameron and J. C. Thomas (Toxicity of Certain Chlorine Derivatives of Benzene, *J. Path. & Bact.* **44:** 281, 1937), who, working with orthodichlorobenzene, observed definite necrosis of the liver in mice twenty-four hours after a twenty-four hour exposure to a concentration of 0.005 per cent. However, the human cataracts that Dr. Berliner describes are more likely due to metabolic changes than to the coexisting dysfunction of the liver. Disorders of the liver, though clinically common, have not been significantly associated with cataract; nor have animals that I have observed after experimentation on the liver shown any related changes in the lens.

Despite exhaustive research on naphthalene cataracts in rabbits, the mechanisms involved remain almost as mysterious as ever. None of the derivatives of naphthalene is similarly effective. In a letter recently received, Dr. Cecil K. Drinker, professor of physiology at Harvard Medical School, stated: "On one occasion we did get a cataract in two rats exposed to a specially made chlorinated naphthalene, but this instance was one out of exposure to similar compounds of many hundred animals, and I do not regard it as significant." Cataract produced by a definite chemical is not the simple problem one might assume. Why does alpha dinitrophenol produce cataract in man but not in animals, or why does thallium acetate produce cataract in rats but atrophy of the optic nerve in man?

Paradichlorobenzene is cheap, selling at 25 to 50 cents per pound. Since 1918 it is steadily displacing naphthalene, hydrogen cyanide and carbon bisulfide as a disinfesting agent. The odor is offensive, the action is rapid, and it is not a fire hazard. As an insecticide, the fumes are fatal to larvae and pupae, and a highly saturated atmosphere will also kill the eggs in two weeks. Since it is difficult to take adequate precautions against the fumes, the British Ministry of Health in 1936 warned against its use for the disinfestation of inhabited houses. The application of paradichlorobenzene to growing plants is likewise undesirable. Public interest requires that the sale and use of this chemical be properly safeguarded. If distributors of the product recognize their responsibility to the consumer, they should voluntarily and without delay modify their present labels in accordance with Dr. Berliner's suggestions.

IRIDENCLEISIS IN GLAUCOMA

EVERETT L. GOAR, M.D.
AND
JACOB F. SCHULTZ, M.D.
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The fact that so many operations have been described for the relief of ocular hypertension indicates that none are entirely satisfactory. The procedure we have used for several years is not original, but as it is not widely used we feel that our experience with it is worth recording. After several unsatisfactory experiences with other kinds of filtering operations, some ten years ago one of us adopted iridotasis for certain types of glaucoma. This was satisfactory so far as the relief of hypertension was concerned, but the updrawn pupil caused a rather unsightly eye. For a number of years then we used iridencleisis through the usual keratome incision. Occasionally when the anterior chamber was shallow we scratched the anterior lens capsule with the lance point. While this does not necessarily produce a cataract, it may do so, and it often produces monocular diplopia. This accident happens occasionally in the hands of the most skilful surgeon, but it can be avoided by the use of a different incision from the usual one.

Critchet,[1] of London, performed the first intentional iris inclusion operation in 1858. A year later Coccius,[1] of Berlin, combined iridectomy with inclusion of a pillar of iris in the wound. However, the operation never became popular and was soon abandoned from fear of irritation to the iris and possible infection. Herbert [2] and Bader [3] later produced prolapse of the iris with iridectomy and appreciated its value in reducing tension, but the report of the results in Holth's [4] series of cases, pub-

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Read before the Section on Ophthalmology at the Ninetieth Annual Session of the American Medical Association, St. Louis, May 17, 1939.

1. Cited by Pischel, K.: Glaucoma: An Historical Review, Am. J. Ophth. **11**:789, 1928.

2. Herbert, H.: Subconjunctival Fistula Formation in the Treatment of Primary Chronic Glaucoma, Tr. Ophth. Soc. U. Kingdom **23**:324, 1903.

3. Bader, C., in Graefe, A., and Saemisch, T.: Handbuch der gesammten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1874-1880, vol. 3, p. 363; Sclerotomy in Glaucoma, Tr. Internat. M. Cong., London **3**:98, 1881.

4. Holth, S.: Ein neues Prinzip der operativen Behandlung des Glaukoms (Fistulasubconjunctivalis camerae anterioris), Ber. ü. d. Versamml. d. ophth. Gesellsch. (1906), 1907, p. 123.

lished in 1906, did more to popularize the operation than any previous article. Many authors have described their results in recent years, but still the operation has not attained the place it deserves in ophthalmic surgery. Wolfe [5] did the operation on 4 eyes in which glaucoma developed following cataract extraction, with satisfactory results. Gifford [6] reported its use on 5 buphthalmic eyes, with satisfactory results in 3 eyes after one operation. The fourth eye required a second operation, and the tension in the fifth was controlled, but the vision failed after two years. Pillat [7] reported that 87 per cent of 100 patients were cured by iridencleisis. Gjessing [8] in 1923 reported on 61 operations; 80 per cent of the patients had normal tension and unaltered vision and 77 per cent had preservation of the visual fields. His later reports on a larger series of operations gave essentially the same figures. Holth [9] claimed that 85 per cent of his operations were successful. Constantine [10] reported satisfactory results in 95 of 107 eyes and stated that he believed that in none of his cases was loss of vision due to the operation. Wilmer [11] recorded successful results in 87.4 per cent of 517 operations. Holst [12] operated on 534 eyes, with successful results in 83.6 per cent in regard to vision, in 96.6 per cent relative to the visual fields and in 90.8 per cent relative to tension. Hagen [13] reported on iridencleisis in 81 cases, with normal tension in 75 per cent; by the aid of miotics after operation in certain cases tension was reduced to normal in 93.8 per cent. Blaickner [14] reported normal tension after 90 per cent of 56 operations.

The series of operations which we wish to report is not large, but practically all the patients were from private practice and we have been

5. Wolfe, O. R.: Iridencleisis for Glaucoma with Aphakia, Am. J. Ophth. **14**:769, 1931.

6. Gifford, S. R.: Iridencleisis in Buphthalmos, Arch. Ophth. **11**:751 (May) 1934.

7. Pillat, A.: Die Fillrationsnarbenbildenden Glaukomoperation mit besonderer Berücksichtigung der Iridencleisis antiglaucomatosa Holth, Ztschr. f. Augenh. supp., 1928, p. 1.

8. Gjessing, H. G. A.: Holth's Iridencleisis Antiglaucomatosa: Follow-Up Examination of One Hundred and Twenty-Two Eyes from Six to One Hundred and Fifty-Nine Months After Operation, Arch. Ophth. **6**:489 (Oct.) 1931.

9. Holth, S.: Iridencleisis antiglaucomatosa, Ann. d'ocul. **137**:345, 1907.

10. Constantine, K. W.: Lundgaard's Modification of Holth's Iridencleisis, Am. J. Ophth. **20**:728, 1937.

11. Wilmer, W. H., and others: Discussion on Results of Operative Treatment of Glaucoma, Tr. Ophth. Soc. U. Kingdom **47**:230, 1927.

12. Holst, J. C.: Results of Iridencleisis in the Years 1928 to 1932, Acta ophth. **12**:348, 1934.

13. Hagen, S.: Comparative Value of Holth's Tangential Sclerectomy and Iridencleisis, Acta ophth. **10**:88, 1932.

14. Blaickner, J.: Ueber Iridenkleisisoperationen, Ztschr. f. Augenh. **72**:265, 1930.

able to follow them closely. We have never felt that patients who maintain vision, visual fields and normal tension under miotics should be operated on provided they can be closely observed and are cooperative. This attitude is not conducive to a large series.

We have operated on 72 eyes in 52 patients, and they have been observed from two to one hundred and eighteen months. Tension was reduced to normal in 96 per cent, although miotics were used in a few of the cases. Vision was improved in 25 per cent, remained stationary in 63 per cent and decreased in 12 per cent. Visual fields improved in 20 per cent, were stationary in 69 per cent and decreased in 11 per cent. The series includes 1 case of bilateral buphthalmos in which operation was done on Nov. 23, 1938. To date the result in each eye has been satisfactory, although it has been a relatively short period since the operation.

The principle of all iris inclusion operations is to form a permanent drainage route from the anterior chamber to the subconjunctival tissue, from which the aqueous may be absorbed. This is accomplished by using a portion of iris tissue as a seton. Herbert [15] and Collins [16] found that all cystoid scars examined after iridectomy contained iris tissue. Holth [17] examined 6 eyes histologically and found that all had fistulas lined with pigment epithelium from the iris and that the bulbar conjunctiva covering the wound had normal epithelium. He observed that the conjunctival connective tissue fibers were somewhat closer together and richer in cells than normal, especially in that area toward the episcleral tissue from which the conjunctiva was loosened at operation. None of the cells showed degenerative changes.

There are many modifications of iridencleisis. It seems that each author has a slightly different method of arriving at the same end. Those who are interested may consult the articles of Holth,[18] Greenwood,[19] Gifford,[20] Jervey,[21] Ziporkes,[22] Constantine,[10] Wille,[23] Horner,[24] del Barrio,[25] Roeder [26] and others.

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15. Herbert, H.: The Future of Iris Inclusion in Glaucoma, Brit. J. Ophth. **14**:433, 1930.

16. Collins, in discussion on Herbert.[15]

17. Holth, S.: Anatomical Examination of Six New Cases of Subconjunctival Fistula Scars from Five to Seventy-Two Months After Successful Iridencleisis or Limbal Sclerectomies in Chronic Glaucoma, Brit. J. Ophth. **6**:10, 1922.

18. Holth, S.: New Technic in Punch Forceps Sclerectomy for Chronic Glaucoma: Tangential and Extralimbal Iridencleisis Operations Epitomized 1915 to 1919, Brit. J. Ophth. **5**:544, 1921.

19. Greenwood, A.: Combined Iridencleisis and Sclerectomy for Chronic Glaucoma, Am. J. Ophth. **13**:301, 1930.

20. Gifford, S. R.: Iridencleisis with Water Tight Closure of the Conjunctiva, Tr. Am. Acad. Ophth. **33**:117, 1927.

(Footnotes continued on next page)

TECHNIC

After trying various technics, we adopted the present one several years ago as the safest and most satisfactory from all standpoints. If the patient has vascular hypertension, we give 50 cc. of a 50 per cent sucrose solution thirty minutes before operation. Miotics are discontinued at least twelve hours before operation. Instillation anesthesia of cocaine and epinephrine hydrochloride is used, and a retrobulbar injection of procaine hydrochloride containing a few drops of epinephrine hydrochloride is made just prior to operation. A large bleb is made above the upper limbus with procaine hydrochloride, and a generous flap including the whole thickness of the conjunctiva is dissected down to the limbus. If there is much bleeding, a pledget of cotton soaked with epinephrine hydrochloride is tucked under the flap and left as long as necessary. With the point of a keratome, the sclera is scratched through layer by layer 1 mm. behind the lower edge of the flap for a distance of about 6 mm., in the manner Elschnig described for "iridectomia ab externo." When aqueous begins to escape the scratching is carried on gently until the fibers are cut through the whole length of the incision. It is neither necessary nor desirable to use scissors to enlarge the incision. A little pressure on the upper lip of the wound will usually spring the iris into it. The iris is pulled out to expose the pigmented margin, and a meridional cut is made from the pupillary to the ciliary border. One pillar is held in the forceps and flattened out along the sclera with the pigment layer exposed while the other slips back into the anterior chamber. If there are dense peripheral synechia, it is necessary to separate them before the iris can be delivered. The flap is replaced and closed with a few sutures. Atropine is instilled, and the eye is covered. Gentle massage is started the second day and continued several weeks.

Iridencleisis is especially useful in cases of glaucoma simplex, as an interval operation between attacks of acute glaucoma and in cases of that type of glaucoma classed as subacute, in which the tension is high even though miotics are used consistently. During acute attacks and in cases of chronic congestive glaucoma it is not the operation of choice, and it is not efficient in cases of secondary glaucoma due to occlusion of the central vein of the retina.

COMPLICATIONS

Complications due to the operation are remarkably few. Retinal hemorrhages may occur in persons with sclerotic vessels, as in any other

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21. Jervey, J. W.: Sclero-Post-Iridectomy, a Rational Operation in Glaucoma, Tr. Am. Ophth. Soc. **25**:160, 1927.

22. Ziporkes, J.: Modification of the Iridencleisis Technic, Arch. Ophth. **19**: 583 (April) 1938.

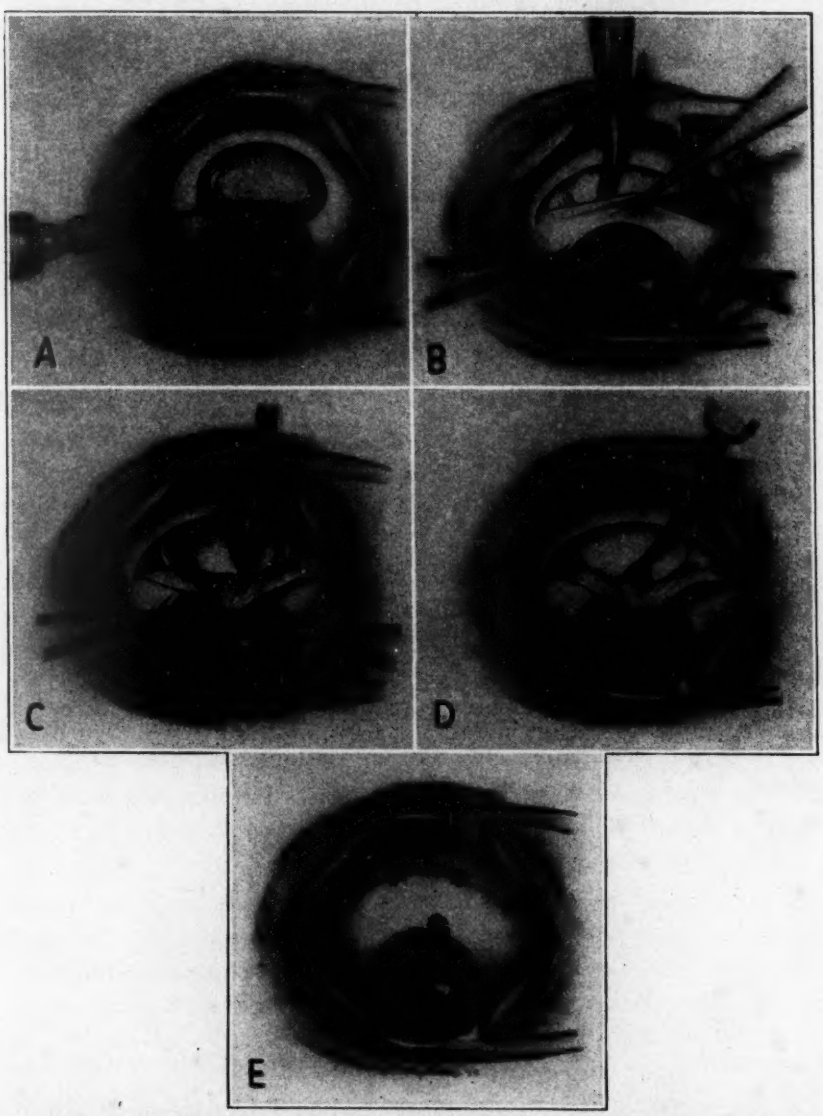
23. Wille, W. A.: Sclerectomy with Iridencleisis, Brit. J. Ophth. **20**:229 (April) 1936.

24. Horner, W. D.: Treatment of Iris Bombé by Iridectomia ab Externo: Report of Cases, Arch. Ophth. **15**:70 (Jan.) 1936.

25. del Barrio, A.: Internal Fistualization by Sclerociliary Iridencleisis in Glaucoma, Ann. d'ocul. **171**:977, 1934.

26. Roeder, J. G.: Cyclodialysis and Subscleral Iridencleisis, Acta ophth. **6**:390, 1928.

type of decompression operation. This has not occurred since we have adopted the use of sucrose solution by intravenous injection prior to operation in selected cases, but we have not used this long enough to be sure of its value. We have had only one cataract develop as a result



Technic of operation. *A* shows the injection for bleb formation; *B*, dissection of the flap; *C*, the scratch incision; *D*, delivery of the iris, and *E*, the completed operation.

of the operation, and this may have been due to an attempt at massaging blood from the anterior chamber, although the opacities began in the posterior cortex. In another patient the scratch incision was made too

far back, and blood seeped into the anterior vitreous. This has absorbed slowly. Uveitis is an occasional complication, and it has always yielded to treatment when the patient could be observed properly after operation. It must be remembered that opacities of the lens existing prior to operation will often be uncovered by the coloboma, and vision will seem to be diminished unless it is taken with a pinhole disk.

SUMMARY

The advantages of this operation have been mentioned by other authors, especially Butler [27] and Horner.[24] We believe they might be summed up as follows:

1. The operation disturbs the eye little, and seldom is there appreciable iritis.

2. Usually little change in refraction is produced.

3. The anterior chamber is quickly reformed, and there is no prolonged hypotony. In fact, the tension may be a trifle elevated for awhile, so that miotics may be necessary for a few weeks.

4. The filtering scar usually is permanent.

5. Late infection is rare. In a large series of cases Erola [28] found that it was about one fifth as frequent as following the trephine operation. We have had several patients who contracted acute conjunctivitis after iridencleisis without intraocular infection.

6. A shallow anterior chamber does not make the operation more difficult.

7. The anterior chamber can be emptied slowly. It is usually unnecessary for any instrument to enter the anterior chamber.

8. The scratch incision enters the anterior chamber much closer to the base of the iris than the conventional keratome incision. Scratching or rupture of the anterior lens capsule does not occur.

We recommend iridencleisis by scratch incision as a safe and efficient operation for the types of glaucoma for which it is adapted. It is useful for the occasional operator as well as for the ophthalmic surgeon of large experience. It merits greater prominence than it has received.

ABSTRACT OF DISCUSSION

Dr. Clyde A. Clapp, Baltimore: I should like to emphasize one point in the history of this operation as mentioned by the essayists. In

27. Butler, T. H.: Iridencleisis and Trap-Door Iridectomy in the Treatment of Glaucoma, Brit. J. Ophth. **16**:741, 1932.

28. Erola, A.: Frequency of Late Infection After Elliots' Trephine and Holth's Iridencleisis, Acta ophth. **12**:137, 1934.

1857 Critchet published an article in the first number of the *Royal London Ophthalmic Hospital Reports* describing an operation called iridotasis. He further stated that if after prolapsing the iris into the wound too much bulging occurred, he excised the excess iris and left the pillars of the iris in the wound. This then practically constituted iridencleisis.

He also discussed the operation of iridectomy which von Graefe had earlier in 1857 described in the third volume of the *Archives für Ophthalmologie*. Von Graefe believed that the lowered tension resulting from his iridectomy was obtained by the restoration of the free communication between the anterior and the posterior chamber, while Critchet attributed the results of the iris inclusion operation to the reestablishment of a normal balance.

In connection with this discussion it is interesting to note that von Graefe had been a former pupil of Critchet.

The authors have mentioned that Dr. Wilmer recorded 517 cases in which iris inclusion was done, with successful results in 87.4 per cent. I wish to call attention to the fact that these cases were collected from the literature and were not his own, for at that time (1927) he had not performed the operation. He was persuaded with difficulty by Dr. Walsh to use it, and then only after Dr. Walsh had seen some of my patients at the Wilmer Ophthalmalogical Institute for whom the results were successful.

The technic which the authors have described of entering the anterior chamber from without in those eyes with shallow anterior chambers is to be commended, but in the ordinary eye with a shallow chamber the incision can be safely performed without danger of touching the capsule of the lens by using a narrow Graefe knife and always keeping the point of the knife in front of iris tissue. The authors have omitted to mention the complication most feared, the one which seldom occurs and also the one that has received the most criticism, namely, sympathetic ophthalmia. Those who remember the first reports on this operation before society meetings will remember the caustic criticisms that were applied to this unsurgical procedure. However, a few cases of sympathetic ophthalmia have been reported. Olsson reported 1 case of sympathetic ophthalmia which occurred two months after iridencleisis and another case previously reported by Herbert, but he also found 6 cases which occurred following an Elliot trephine operation.

Herbert obtained successful results in 93.2 per cent of cases of chronic glaucoma, in 83.3 per cent of cases of acute glaucoma and in 50 per cent of cases of complicated glaucoma, or in 90 per cent of all the cases in which this method was used. The authors' successful results in 96 per cent of their cases compares most favorably.

I have not seen any late infections in my own cases, and I have obtained satisfactory reduction in tension in the cases in which there are no complications. Expulsive hemorrhage occurred in a man of 78 who had had absolute glaucoma for several years and who had extensive cardiovascular disease.

Colonel Wright stated that the operation has practically been abandoned in his clinic in India because it seems necessary to follow up the operation with massage, and this is impracticable for his patients. Other surgeons feel that iridencleisis should be used only in cases of simple chronic glaucoma. I have used the method for all types of glaucoma, and the results have convinced me that while possibly it is unsurgical it gives better results than any operation so far devised.

Dr. Thomas D. Allen, Chicago: Recently I reviewed the histories of 40 cases in which this operation was done either by Dr. William H. Wilder or by myself. The results have not all been successful. In some cases the operation was done as a secondary procedure and in some as a primary procedure. The successes usually followed meticulous care in the details of the operation, and as some articles have appeared in recent books which left much to be desired in the way of description of the technic, I wish to reemphasize what Dr. Wilder stressed.

1. As to preliminary preparations, the tension should be lowered for several days or weeks if possible to below 40 mm. (Schiötz) and the congestive aspects of the acute varieties lessened as much as possible by miotics and if necessary by posterior sclerotomy.

2. The incision in the conjunctiva should be 8 mm. from the limbus and should come down on the sclera just anterior to the superior rectus muscle. It should be nearly horizontal and not carried downward to the limbus on each side.

3. As thick a flap as possible should be obtained with the least possible dissection; that means to hug the sclera to within 2 mm. from the limbus. Infiltration of conjunctiva in the area of operation often interferes with this. We therefore infiltrate elsewhere.

4. The hemorrhage is stopped before the anterior chamber is entered. A barely glowing cautery tip will do this if it cannot be done by pressure.

5. The chamber is entered obliquely immediately anterior to the plane of the iris. (One can then cut off the anterior scleral lip if desired.)

6. The iris is grasped at the pupillary margin and withdrawn, and a small cut is made at the pupillary margin at one side of the forceps.

7. The iris limb held in the forceps is twisted so that the pigment surface is rolled on itself like a funnel, and the other limb is allowed to slide back into the chamber.

8. The chamber is irrigated if blood of any consequence appears. The wound should not be closed if blood is present in the chamber. (If it appears the next day, it may be left; it usually disappears spontaneously within a few days.)

9. The conjunctiva may be sutured if necessary.

10. A light bandage is applied for two or three days.

11. One drop of a solution of atropine is usually instilled once at the first dressing.

12. The patient may be allowed up after twenty-four to forty-eight hours.

The more nearly one holds to this technic, the less one will regret operating for glaucoma.

DR. ALLEN GREENWOOD, Boston: I have advocated in several papers that in addition to iridencleisis operators should cut out a little of the scleral lip. When the small lip of sclera is seen as the conjunctiva is pulled forward, a bit of that sclera is nipped off, with either a punch or a pair of scissors. The necessity of massage on such eyes is eliminated; a good filtering cicatrix will be obtained in practically every case.

In regard to the formation of cataract after an operation for glaucoma, after Dr. Goar and his associate have watched their patients for twenty years they will be surprised to find in how many of them cataracts will develop. I have had only 1 case in which cataract developed immediately after iridencleisis. I have had 1 case in which sympathetic ophthalmia developed three years after the operation. I have not observed any cases of late infection.

DR. JOHN GREEN, St. Louis: I agree with Drs. Goar and Schultz that a large flap is desirable. To insure a large thick flap, i. e., one that includes the subconjunctival tissue, a generous quantity of procaine hydrochloride can be injected under the conjunctiva. It is important to get down to the scleral level with the first snip of the scissors and to maintain the dissection at that level.

For the incision, I prefer the knife designed by Lundsgaard. The model I use has a narrow cutting blade 10 mm. long with the shaft set at a 30 degree angle. With a gentle sawing motion, one "whittles" into the chamber.

I have noted a slow restoration of the anterior chamber. This has never resulted harmfully; possibly it is due to too wide an incision with this type of knife.

I have seen 1 case in which sympathetic ophthalmia followed iridencleisis. The operation was expertly performed. On reviewing the history, it seemed probable that the patient did not have simple glaucoma but an increase of tension due to a low grade uveitis.

One advantage of iridencleisis over trephining is that the filtering pad is flatter and less conspicuous. Thus it is possible to place the incision almost anywhere. On a number of occasions, in dealing with glaucoma complicated by cataract I have placed the flap and incision down and temporally. Thus the subsequent incision for cataract extraction was completely away from the site of the prior incision.

DR. M. GOLDENBURG, Chicago: It is gratifying to see the iris incarceration operation coming to the fore again. I have used the operation for over twenty years. My first report on it included the results in 105 cases of various types of glaucoma. Although I referred to the operation as iridotasis, it has been only slightly modified and is called iridencleisis.

The advantages of the operation have been enumerated and are lasting. I have many such cases under observation at the present time in which iridectomy has been performed on one eye and an operation to prolapse the iris on the other eye; and in some trephining has been done on one eye and an iris inclusion operation on the other.

The success of this operation over a long period of time is dependent from a technical standpoint on two factors: The first is based on the manner in which the conjunctiva is dissected from the underlying sclera. If the incision is made through the conjunctiva directly up to the sclera, one need hardly dissect at all. However, whether one makes a horizontal incision or a similar one, the essential factor is minimum trauma.

The second factor pertains to placing the limbus incision as near to the level of the anterior surface of the iris as possible. When that is done there is no scleral elevation over which the iris must ride. This has been demonstrated in many microscopic specimens.

There are two physical factors, however, which influence the result. If the conjunctiva is very thin, as it is in thin elderly women, an extremely thin flap will be obtained, and it may tear or adhere to the underlying sclera and defeat the primary purpose, namely, drainage. Also, if the iris is very thin, a similar result will follow, but if both the conjunctiva and the iris are comparatively normal, one will get a good result which will last over a longer period than that obtained by any other procedure.

Dr. Sanford Gifford, Chicago: I want to mention two practical points. One is the use of my father's toothed fixation forceps with a clamp to grasp the superior rectus tendon through the intact conjunctiva in order to hold the eye down during the whole course of operation. One grasps the tendon as high as one can in order to make as large a flap as possible, then applies the clamp. It will hold the eye down until one has completed the operation. There is not the danger there would be in an operation for cataract because the incision is so small. When one finishes the operation, a running conjunctival suture patterned after Foster Moore's so-called water-tight suture is used. It is not tied and is much easier to remove than the interrupted sutures which Dr. Schultz showed.

I should also like to speak of the use of this operation in cases of buphthalmos, since Dr. Clapp told me he thought I was exceedingly—plain lucky, as he put it. I suppose I have been, and I might say that I have had another case since in which the operation failed and another case in which it was successful. In the latter case a simple iridencleisis was done on a young baby as soon as a diagnosis was made. In the case in which failure occurred the patient, a child, had an associated congenital anomaly of the lens and the eye was already quite large when it was operated on. However, I think it is a useful operation for buphthalmos. The indication I have for choosing between iridencleisis and trephining is simple. The indication for both exists in chronic simple glaucoma, but when the tension cannot be brought down below 40 or 45 with miotics, I feel that one is a little more likely to get a complete reduction of tension by trephining. When the tension can be brought down below 40 or 45 with miotics, I think that the effect of iridencleisis will usually be sufficient. It may have to be followed by massage.

Dr. John O. McReynolds, Dallas: I should like to emphasize a few points that have already been presented. The procedure as outlined by Dr. Wilder is beyond criticism, and as was suggested by Dr. Allen Greenwood, punching out a portion of the sclera is eminently the thing to do. In my own experience the procedure I have employed is this: Instead of making any kind of flap, I proceed regularly by operating

beneath a broad, undivided conjunctival bridge. The region of the limbus can be reached with perfect satisfaction in that way, and then when the operation is finished one is sure to get the quickest possible result. One does not want to have any inflammatory reaction beyond that which is absolutely necessary. In other words, a quick, clean healing process will give the best final results.

Dr. Jacob F. Schultz, Houston, Texas: Sympathetic ophthalmia was not mentioned in this paper, because only those complications which occurred in our series of cases are included. I am glad it was suggested, because it is a potential danger and it has been mentioned many times in the literature.

RELATIVE SIZES OF OCULAR IMAGES OF THE TWO EYES IN ASYMMETRIC CONVERGENCE

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Differences in size between the retinal images of the eyes may arise in asymmetric convergence because the object fixated will be at a different distance from the two eyes.[1] The magnitude of the difference will obviously increase with the nearness of the object and with the degree of the lateral turning of the eyes. In the normal use of the eyes when looking sideways at near objects, retinal images of objects subtending unequal visual angles to the two eyes are fused into a single composite image. No apparent difficulties arise in this act.

Difficulties may arise, however, when it is necessary to fuse images of unequal size produced artificially with size or power lenses or when patterns of unequal size are presented in the stereoscope. Fusion may not then be possible if the difference in size is outside fusional areas (so-called Panum's areas of sensation[2]). However, apart from the

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In another paper published under the same title (Herzau, W., and Ogle, K. N.: Ueber den Grössenunterschied der Bilder beider Augen bei asymmetrischer Konvergenz und seine Bedeutung für das zweiäugige Sehen. Ein Beitrag zur "Aniseikonia-Forschung," Arch. f. Ophth. **137**:327, 1937) the problem of the relative sizes of the ocular images when the eyes are turned in asymmetric convergence was considered with special reference to the apparent frontal plane (horopter) as supplemented by experiments on the haploscope. The present paper serves a twofold purpose: first, in presenting the problem from a different point of view with complementary and new experimentation and, second, in acquainting readers to whom the article in German is not available with the essential phenomenon involved.

1. This fact was first considered by Desaguliers in 1717 (Desaguliers, J. T., cited by von Rohr, M.: Zur Kenntnis älterer Ansichten über das beidäugige Sehen, Ztschr. f. Instrumentenk. **36**:226, 1916) and later verified by C. Wheatstone (Contribution to the Physiology of Vision: I. On Some Remarkable, and Hitherto Unobserved, Phenomena of Binocular Vision, Phil. Tr., London, 1838, p. 371).

2. Tschermak, A.: Optischer Raumsinn, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1930, vol. 12, pt. 2, p. 894.

Fusion may persist in the central parts of the binocular visual field with a given difference in the sizes of the images when it is not possible in the more peripheral regions. This is because the angular extent of Panum's areas near the foveas is large, relative to the angular extent of a small difference in size, whereas the reverse becomes true in the periphery (Ames, A., Jr., and Ogle, K. N.: Size

consideration of fusion difficulties, false stereoscopic depth values usually arise in binocular vision when images of different size have to be fused.[3] These false space perceptions may in themselves be a source of visual disturbances.[4] In this respect, evidence which has accumulated shows that the correction of aniseikonia with suitable lenses [5] is capable of removing certain subjective disorders, such as headaches, photophobia and dizziness, which have existed despite the fact that no anomalies of refraction or muscle balance are present or which persist after a refractive or prismatic correction has been given.[6] Changes in binocular depth perception caused by introducing a difference in the sizes of the images is evidence of a functional importance of the relative sizes of the ocular images. At the same time the large amount of clinical experience establishes evidence of the importance of correcting existing differences in size.

However, one is confronted with the problem: If such differences in size may cause serious functional disturbances, how can the differences associated with high degrees of asymmetric convergence be tolerated, even apart from the fact that normally one does not use the eyes in asymmetric convergence for any sustained period of time?[7]

The magnitude of the difference in the sizes of the retinal images that would occur in asymmetric convergence because of the difference in distance from the object to the two eyes (the geometric difference) can be computed from $R = \sqrt{\frac{1 + A \sin\beta}{1 - A \sin\beta}}$ in which $A = 2ab/(a^2 + b^2)$; R, the ratio of the sizes of the images (the image of the left eye to that of the right eye); b, the visual

and Shape of Ocular Images: III. Visual Sensitivity to Differences in the Relative Size of the Ocular Images of the Two Eyes, Arch. Ophth. **7**:904 [June] 1932).

3. Ames, A., Jr.; Ogle, K. N., and Gliddon, G. H.: Corresponding Retinal Points, the Horopter and Size and Shape of Ocular Images, J. Optic. Soc. America **22**:538, 1932. Ogle, K. N.: The Induced Size Effect: I. A New Phenomenon in Binocular Vision Associated with the Relative Sizes of the Ocular Images of the Two Eyes, Arch. Ophth. **20**:604 (Oct.) 1938.

4. Ames, A., Jr.: Aniseikonia: A Factor in the Functioning of Vision, Am. J. Ophth. **18**:1014, 1935; in discussion on Jackson, E.: Importance of Aniseikonia, Tr. Sect. Ophth., A. M. A., 1936, p. 34.

5. Ogle, K. N.: The Correction of Aniseikonia with Ophthalmic Lenses, J. Optic. Soc. America **26**:323, 1936.

6. (*a*) Carleton, E. H., and Madigan, L. F.: Size and Shape of Ocular Images: II. Clinical Significance, Arch. Ophth. **7**:720 (May) 1932. (*b*) Doane, H. C.: The Clinical Significance of Differences in the Relative Size and Shape of Ocular Images, Am. J. Optometry **11**:390, 1934. (*c*) Hughes, W. L.: Aniseikonia: Some Clinical Observations, Am. J. Ophth. **18**:607 and 715, 1935; **20**:887, 1937. (*d*) Bielschowsky, A.: Aniseikonia, Acta ophth. **16**:188, 1938.

7. This question was also raised by E. J. Ludvigh: (Aniseikonia, Am. J. Ophth. **19**:292, 1936).

distance; $2a$, the interpupillary distance, and β, the degree of asymmetric convergence (fig. 1). The computed magnitudes of these ratios for various visual distances and for various degrees of asymmetric convergence are given in table 1.[8] In view of the fact that differences in size greater than 5 per cent are rarely found in patients with binocular

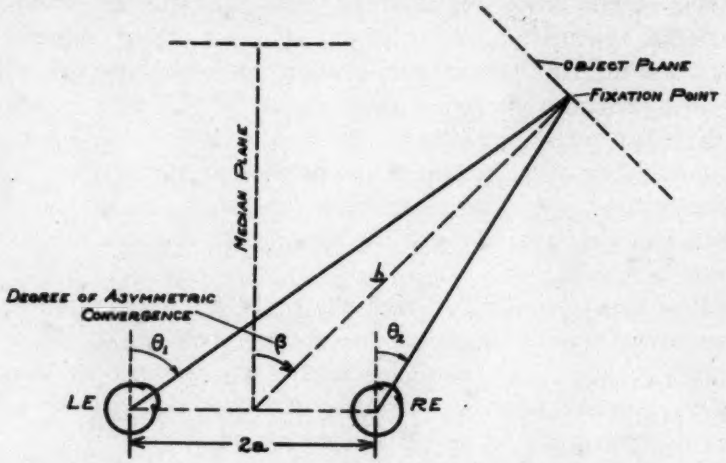

Fig. 1.—Geometric arrangement of the object and the two eyes in asymmetric convergence.

TABLE 1.—*Theoretic Ratios of the Sizes of the Retinal Images with the Eyes in Asymmetric Convergence, Due to the Difference in Distances from the Two Eyes to the Object Observed*

	Computation for an Interpupillary Distance of 65 Mm.			
	Degree of Asymmetric Convergence			
Visual Distance	10°	20°	30°	40°
6 meters	1.002 (0.2%)	1.003 (0.3%)	1.004 (0.4%)	1.006 (0.6%)
75 cm.	1.015 (1.5%)	1.030 (3.0%)	1.044 (4.4%)	1.057 (5.7%)
40 cm.	1.028 (2.8%)	1.056 (5.6%)	1.084 (8.4%)	1.109 (10.9%)
33 cm.	1.033 (3.3%)	1.069 (6.9%)	1.102 (10.2%)	1.134 (13.4%)
20 cm.	1.054 (5.4%)	1.114 (11.4%)	1.173 (17.3%)	1.229 (22.9%)

vision, save when the fusion is deficient or when binocular vision is impaired, it becomes clear that in some instances the geometric difference

8. These values are strictly correct only in the vertical meridian of the two eyes, for in the horizontal meridian the geometric difference in size depends also on the position of the object, in the sense of a rotation about a vertical axis at the fixation point. If the face of the object observed remains normal to the line joining the fixation point on the object and the midpoint of the interpupillary base line, then the geometric difference in size will be approximately the same in all meridians and of the amount given in the table.

in size ought to interfere with binocular vision when the eyes are turned into asymmetric convergence.

The implications that might be raised by this discussion have not been overlooked, for as early as 1930 preliminary studies had been made on this problem. The measurements made even at that time with usual size instruments suggested that the ocular image of the eye farther from an obliquely viewed object becomes larger or that of the other eye smaller,[9] thus compensating for the difference in distance.

EXPERIMENTATION

Of the different ways in which the relative sizes of the ocular images in asymmetric convergence might be measured, the haploscope obviously offers a simple method. It may be described as follows (fig. 2): With the head of the observer adjusted accurately in the instrument, two arms are so arranged as to turn about vertical axes through the centers

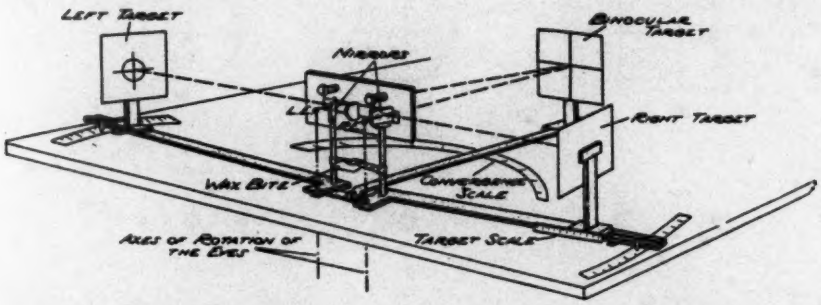

Fig. 2.—Schematic drawing of the haploscope as used to determine the apparent change in sizes of the ocular images when the eyes turn in asymmetric convergence.

of rotation of the two eyes. On these arms, half-silvered mirrors are mounted so that the reflected images of appropriate targets at L and R farther out on the arms are seen by the observer as a single target in front. When the two eyes of the observer are adjusted accurately over the centers of rotation of the arms, the two targets will always remain the same distance from the eyes, regardless of how they are turned. The condition of asymmetric convergence can be obtained by turning the arms specified amounts in the same direction. Under such experimental conditions any change which might occur between the relative sizes of the images with lateral gaze will then be susceptible to observation and measurement. With appropriate lenses and with the corresponding change in the convergence of the arms, the images

9. Ames, A., Jr.; Ogle, K. N., and Gliddon, G. H.: Corresponding Retinal Points, Horopter and Size and Shape of Ocular Images, J. Optic. Soc. America 22:575, 1932. (The statement referred to is on page 614.)

of the targets can be projected to various visual distances. The angular size of each target can be changed either by moving it a small distance along the arm, or better, by using appropriate size lenses before the eyes. The efficacy of the apparatus, of course, depends on the choice of suitable and sensitive target patterns. While a number of different types of patterns were used, only the results obtained with the three illustrated in figure 3 need be described, since these are representative.

Circle and Square Patterns.—The simplest pattern consisted of a square before one eye and a circle before the other eye (fig. 3 *A*), the diameter of the latter and the length of a side of the square being the same (3 cm. for a visual distance of 33 cm.). Fusion is maintained either by means of crossed lines at the centers of the targets or by a

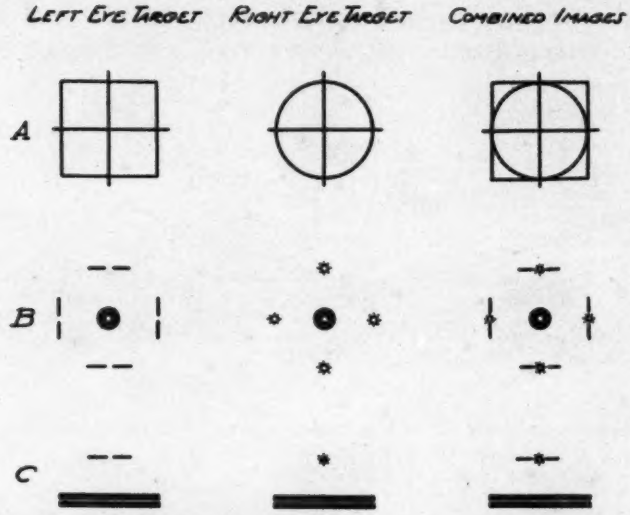

Fig. 3.—Types of haploscope targets used: *A*, circle and square pattern; *B*, eikonic pattern, and *C*, central fixation.

single pair of crossed lines on a third target mounted in front of the observer and seen through the half-silvered mirrors. The circle will appear tangent to the sides of the square if the observer has neither a size difference nor a fixation disparity.[9] If a fixation disparity exists (and it usually does in the horizontal meridian), the circle will appear displaced relative to the square. The relative sizes of the circle and square can be easily compared, however, whether such a displacement exists or not.

The procedure is as follows: With the instrument set for symmetric convergence of the eyes, the angular sizes of the targets are adjusted until the circle and square appear the same size. The two arms are then turned for an asymmetric convergence of the eyes. Any apparent

change that may occur between the relative sizes of the ocular images with the change of the eyes from symmetric to asymmetric convergence will be evident at once.

That an apparent change does occur for near vision is rather a striking experience to one who sees it for the first time. At near vision with free fixation, that is, in ordinary observation where the eyes may be allowed to move freely, all observers have reported that the target before the eye which turns nasally appears relatively larger. This phenomenon is shown schematicallly in figure 4, in which the square and the circle are seen by the left and the right eye, respectively. When the arms are turned for an asymmetric convergence of the eyes to the right, the square appears larger.

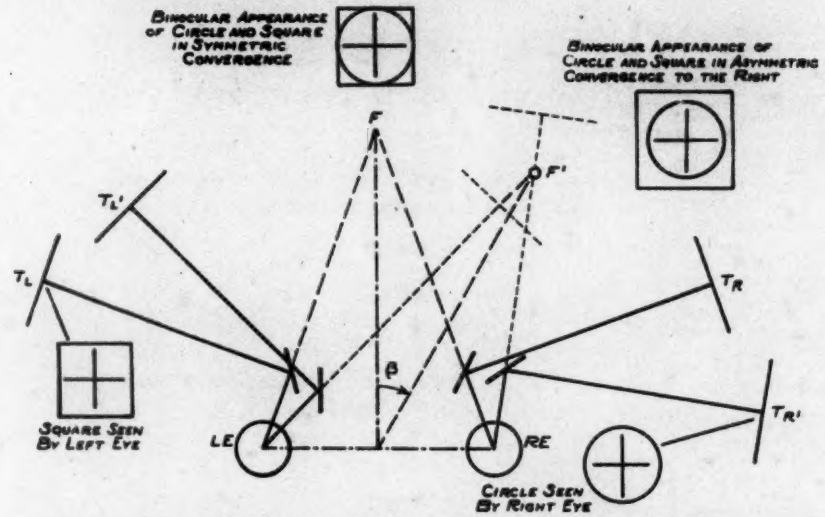

Fig. 4.—Representation of the phenomenon of an apparent change in the relative sizes of the ocular images when the eyes are turned in asymmetric convergence, for the circle and square target patterns.

The magnitude of the difference can be readily determined, either by the amount that one of the targets must be moved along the arms toward or away from the eyes or by the selection of a size lens which, placed before one eye, equalizes the apparent sizes of the patterns. Typical data obtained by one observer are given in table 2 *A* and are illustrated graphically in figure 5. When the relative sizes were determined quantitatively, a larger change was usually found in the vertical than in the horizontal meridian, though several observers obtained the same change in both meridians.

This apparent change in the relative sizes of the images is in the direction compensating for the difference in distances which would

normally occur in an asymmetric convergence of the eyes. In figure 5 a solid line has been drawn to indicate the theoretic change in the sizes of the images which would be necessary for a full compensation for the difference in size when the two eyes are turned in asymmetric convergence. The agreement between the data for the vertical meridian and this theoretic line is striking. All observers reported difficulty in

TABLE 2.—*Data (of K. N. O.) with Circle and Square Targets on the Haploscope, Showing the Apparent Change in the Relative Sizes of the Ocular Images in Asymmetric Convergence**

	Asymmetric Convergence to Left				Symmetric Convergence	Asymmetric Convergence to Right			
	20°	15°	10°	5°		5°	10°	15°	20°
A. Free Fixation †									
Vertical meridian									
Mean setting—cm.	35.3	35.1	34.4	33.9	33.4	33.0	32.6	32.1	31.7
Mean deviation	0.0	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1
Ratio sizes of ocular images L:R	0.944	0.949	0.969	0.983	0.997	1.010	1.022	1.038	1.051
Horizontal meridian									
Mean setting—mm.	33.8	33.8	33.6	33.4	33.0	33.3	33.1	32.8	32.4
Mean deviation	0.2	0.1	0.1	0.1	0.1	0.2	0.1	0.1	0.2
Ratio sizes of ocular images L:R	0.986	0.986	0.992	0.997	1.010	1.000	1.007	1.016	1.029
B. Constant Central Fixation ‡									
Vertical meridian									
Mean setting—mm.	33.9	....	33.9	....	33.4	....	33.0	....	32.7
Mean deviation	0.2	....	0.2	....	0.2	....	0.1	....	0.1
Ratio sizes of ocular images L:R	0.983	....	0.983	....	0.997	....	1.010	....	1.019
Horizontal meridian									
Mean setting—mm.	33.4	....	33.0	....	33.4	....	32.7	....	32.7
Mean deviation	0.0	....	0.0	....	0.2	....	0.1	....	0.1
Ratio sizes of ocular images L:R	0.997	....	1.010	....	0.997	....	1.019	....	1.019
C. Free Fixation §									
Vertical meridian									
Mean setting—cm.	40.0	....	40.0	....	40.0	....	39.6	....	39.6
Mean deviation	0.0	....	0.0	....	0.1	....	0.1	....	0.0
Ratio sizes of ocular images L:R	1.000	....	1.000	....	1.000	....	1.009	....	1.009
Horizontal meridian									
Mean setting—cm.	38.6	....	39.0	....	38.8	....	39.0	....	39.2
Mean deviation	0.1	....	0.0	....	0.0	....	0.0	....	0.1
Ratio sizes of ocular images L:R	1.028	....	1.020	....	1.023	....	1.020	....	1.016

* Apparent sizes were made equal by moving the right target on the arm.
† The visual distance was 33 cm.
‡ The visual distance was 33 cm.
§ The visual distance was 5 meters, obtained by mounting suitable power lenses on the haploscope arms, so as to project the targets at 40 cm. to virtual distances of 5 meters.

making the judgments in the horizontal meridian, due usually to unsteady fixation disparity, but some of them experienced the change in the horizontal meridian in the same direction as that in the vertical, though its magnitude varied with the observer. The data given in figure 5 show only a small change.

Somewhat different results were also found, depending on whether the eyes were allowed to rove freely over the targets or whether a constant fixation was maintained at the centers. In the latter case the comparison of the apparent sizes could be made only with indirect or

peripheral visual judgments. The data given in table 2 *B* and illustrated in figure 6, for the same observer, were obtained with central fixation. The apparent changes with asymmetric convergence are in general much smaller with steady fixation.

When the phenomenon was studied with the instrument adjusted so that the eyes would be adapted for distance vision, little or no

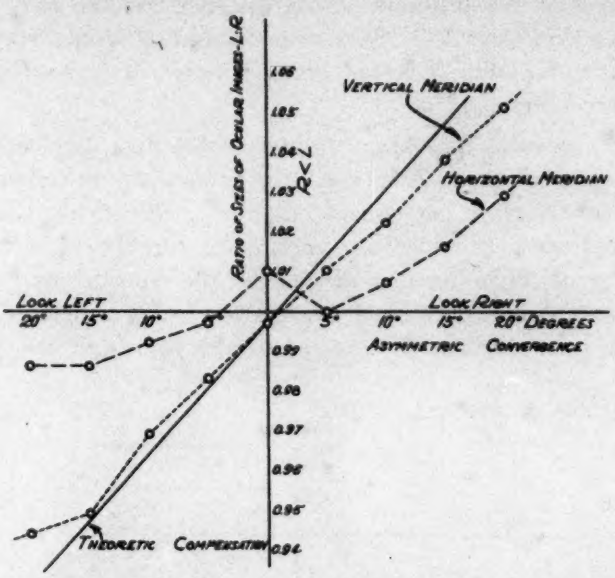

Fig. 5.—Graphic representation of data obtained on the haploscope by K. N. O. with the circle and square patterns with free fixation. The visual distance was 33 cm.

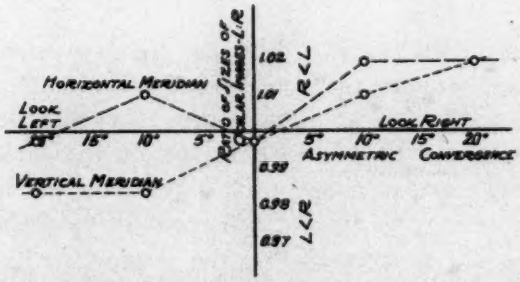

Fig. 6.—Graphic representation of data obtained on the haploscope by K. N. O. with the circle and square target patterns with constant central fixation. The visual distance was 33 cm.

difference between the apparent sizes of the circle and square could be measured when the eyes were turned to asymmetric convergence. For an equivalent visual distance of 5 meters, practically no change could

be measured either with free or with constant central fixation. This is illustrated in the typical set of data given in table 2 *C* and is shown graphically in figure 7.

These results suggest that there may be an adjustment of the relative sizes of the ocular images of the two eyes which offsets or tends to offset the difference in the sizes of the retinal images that would occur because of the different visual distances of the two eyes when in asymmetric convergence. Such a compensation would not be needed with distant vision, but it would become increasingly necessary as the visual distance decreases.

Eikonic Targets.—To obtain more accurate data, haploscopic target patterns similar to those first used for measuring aniseikonia[10] were substituted for the circle and square (fig. 3 *B*). Identical fusion patterns (small rings) were provided for each eye. On the right target four small points of light were arranged in the vertical and horizontal meridians at a visual angle of 4 degrees from the center. On the left target four small broken lines were drawn to match the pattern of

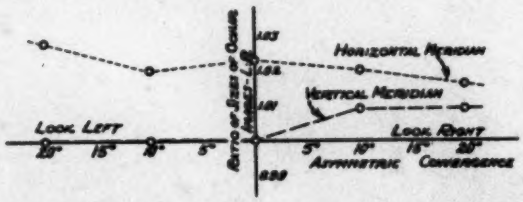

Fig. 7.—Graphic representation of data obtained on the haploscope by K. N. O. with the circle and square target patterns with free fixation. The visual distance was 5 meters.

the lights for the right target. When an observer who had neither a difference in size between the ocular images nor a fixation disparity looked into the haploscope at these targets in symmetric convergence, the lights appeared to lie within the gaps of the broken lines. If, however, the right ocular image was larger than the left, the lights appeared to lie outside the gaps. Such apparent inequality in the sizes of the images could be corrected in the instrument by means of an adjustable size lens system placed before the eyes. Fixation disparity may cause an equal displacement of the light points to one side of their respective lines. The judgments of the relative positions of the lights with respect to the lines will involve small ocular movements, for the observer actually looks over to the lines and lights. Reliance is placed

10. Ames, A., Jr.; Gliddon, G. H., and Ogle, K. N.: Size and Shape of Ocular Images: I. Methods of Determination and Physiologic Significance. Arch. Ophth. 7:576 (April) 1932.

on the fusion of the central figures of the targets to maintain the relative visual directions of the eyes during these ocular excursions. This assumption is based on the general experience that the peripheral fusion stimuli of this order are usually sufficient to control the pointing of the eyes.[11]

The data taken with these targets show small mean variations of differences in size and are, in general, consistent. Typical results obtained when the eyes are turned into asymmetric convergence are given

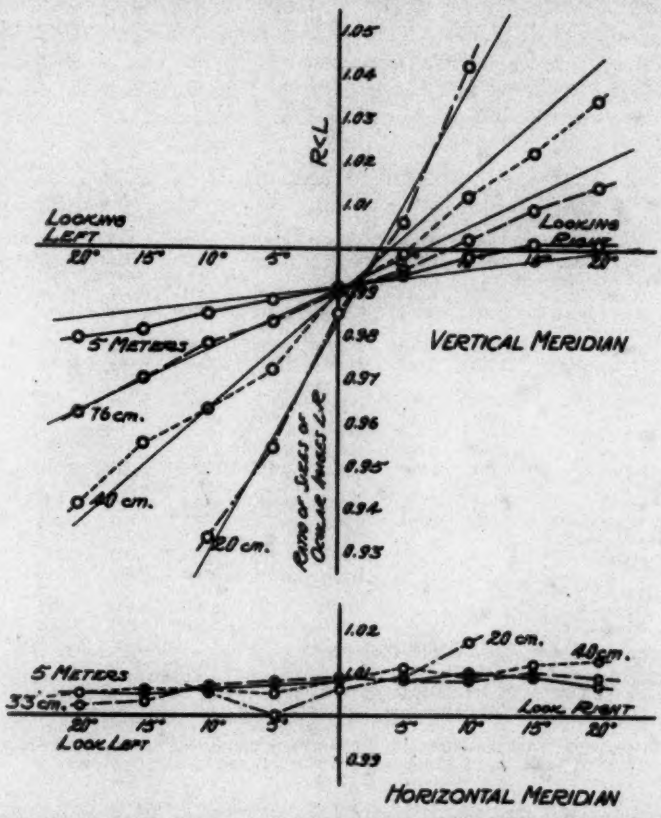

Fig. 8.—Graphic representation of typical data taken on the haploscope with free fixation using eikonic targets, showing the apparent change in the relative sizes of the ocular images in the vertical and horizontal meridians when the eyes turn in asymmetric convergence for several visual distances. The solid lines represent the theoretic change for complete compensation.

in table 3 and are shown graphically in figure 8. The data illustrated have been taken for different visual distances ranging from 20 cm. to

11. Moreover, it is justified by the experiments recently described by Burian, H.: Fusional Movements: The Role of Peripheral Retinal Stimuli, Arch. Ophth. 21:486 (March) 1939.

5 meters. The same compensation phenomenon as already described is found, especially in the vertical meridian. For near visual distances a change appears in the relative sizes of the ocular images, as though the image of the eye which turns nasally were enlarged. This apparent change in size with lateral gaze becomes greater as the visual distance decreases. The narrow lines drawn through the points representing data in the vertical meridian again indicate the theoretic change in size necessary to compensate for the difference in the sizes of the retinal

TABLE 3.—*Data (of K. N. O.) Obtained with Eikonic Targets on the Haploscope, Showing the Change in the Relative Sizes of the Ocular Images of the Eyes in Asymmetric Convergence for Various Visual Distances**

Visual Distance	Meridian	Looking Left				Symmetric Convergence	Looking Right			
		20°	15°	10°	5°		5°	10°	15°	20°
5 meters	Horizontal									
	Mean	1.005	1.005	1.006	1.007	1.008	1.008	1.010	1.009	1.007
	Mean dev.	0.001	0.003	0.001	0.001	0.002	0.003	0.003	0.002	0.001
	Vertical									
	Mean	0.979	0.981	0.985	0.988	0.991	0.994	0.998	1.001	1.001
	Mean dev.	0.002	0.001	0.001	0.001	0.001	0.002	0.001	0.001	0.002
76 cm.	Horizontal									
	Mean	1.002	1.003	1.007	1.008	1.009	1.008	1.008	1.010	1.009
	Mean dev.	0.002	0.002	0.002	0.002	0.001	0.001	0.002	0.001	0.002
	Vertical									
	Mean	0.962	0.970	0.978	0.983	0.990	0.995	1.002	1.009	1.014
	Mean dev.	0.001	0.002	0.001	0.000	0.000	0.001	0.000	0.002	0.000
40 cm.	Horizontal									
	Mean	1.005	1.006	1.006	1.005	1.008	1.011	1.009	1.012	1.013
	Mean dev.	0.002	0.002	0.001	0.001	0.002	0.002	0.002	0.003	0.001
	Vertical									
	Mean	0.941	0.955	0.963	0.972	0.990	0.999	1.012	1.022	1.034
	Mean dev.	0.002	0.002	0.002	0.000	0.001	0.001	0.001	0.000	0.000
20 cm.	Horizontal									
	Mean			1.005	1.000	1.011	1.009	1.017		
	Mean dev.			0.001	0.004	0.002	0.003	0.002		
	Vertical									
	Mean			0.933	0.954	0.985	1.006	1.042		
	Mean dev.			0.002	0.002	0.002	0.002	0.002		
Measurement of Accommodation—Diopters										
40 cm.	Right eye			2.53(0.08)		2.55(0.03)			2.53(0.06)	
	Left eye			2.55(0.11)		2.48(0.04)			2.62(0.02)	

* The relative sizes were measured by an adjustable size lens system and are given here in terms of the ratio of the sizes of the images of the left eye to the right eye.

images due to the difference in distances. These data show remarkable agreement with these theoretic lines. In the horizontal meridian, on the other hand, these particular data (which are representative) show only a slight change in the direction of a compensation. No explanation for this behavior can be made as yet.

These eikonic targets did not permit data to be taken with constant central fixation, since the detail of the lights and lines was too small for their positions to be recognized in peripheral vision.

Similar data for a myope and a presbyope are also illustrated in figures 9 and 10. These show the same change in the relative sizes of

the images when the eyes are turned in asymmetric convergence in the direction which compensates for the difference in distances of the object point from the two eyes.

The particular targets used in this experiment were made so that the state of accommodation of each eye could be determined with the eyes in both symmetric and asymmetric convergence.[12] Point light

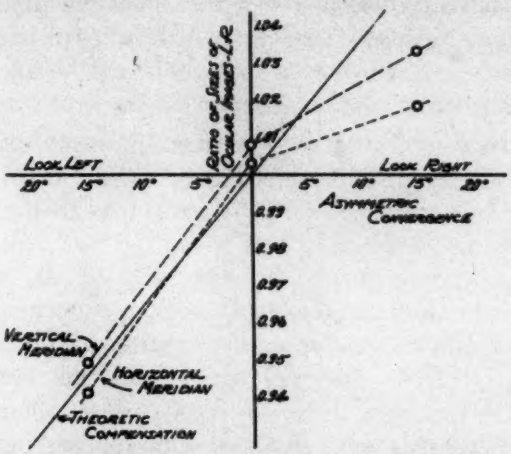

Fig. 9.—Graphic representation of data obtained on the haploscope by H. M. B., a myope, with eikonic targets with free fixation for a visual distance of 40 cm.

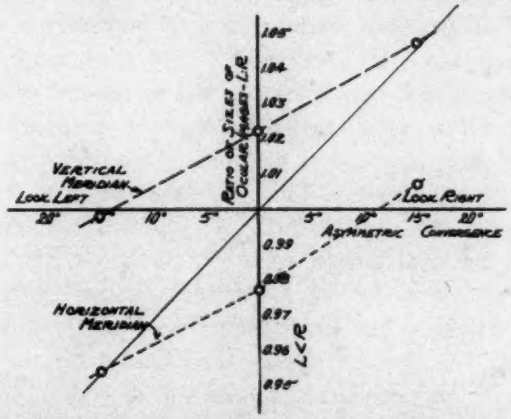

Fig. 10.—Graphic representation of data obtained on the haploscope by A. A. Jr., a presbyope, with eikonic targets with free fixation for a visual distance of 40 cm.

sources were projected by means of suitable lens systems through openings in the center of each of the targets. These points of light could be moved along the arms of the haploscope (and thus along the

12. A description of this method is given in Ames, A., Jr., and Gliddon, G. H.: Ocular Measurements, Tr. Sect. Ophth., A. M. A., 1928, p. 102.

visual axes of the eyes). They could therefore be adjusted by the observer for the sharpest focus. The position of a point image for the sharpest focus would be conjugate to the retina, and the reciprocal of its distance from the eye would be the measure of accommodation (diopters). In table 3 these special data are given for the visual distance of 40 cm. Inspection shows, within 0.25 diopter, little or no change in the state of accommodation of the two eyes when they are turned to asymmetric convergence. Therefore, it is believed that the apparent compensation for the difference in size could not be accounted for on the basis of a differential accommodation of the two eyes.

Peripheral Fusion Pattern.—The measurements made with the eikonic target patterns just described necessitated small ocular movements of about 4 degrees when the judgment as to the displacements of the lights relative to the lines was made. It has been assumed that during those ocular movements the directions of the visual lines of the eyes were controlled by the small fusion patterns at the centers of the targets. Certainly, under such conditions differences in size of a small order (0.1 to 0.3 per cent) can be readily detected. Nevertheless, the fact that different results were obtained when the circle and square patterns were observed with and without ocular movements suggests that the apparent compensatory change in the sizes of the ocular images with asymmetric convergence might be some phenomenon associated with differential ocular movements.

In order to obtain data without ocular movements target patterns as illustrated in figure 3 *C* were used. Two identical horizontal bands were placed at equal distances above or below the centers of the targets. These bands served as peripheral fusion stimuli. A small dot was placed at the center of one target and a short horizontal broken line at the center of the other target. This dot and line device was fixated binocularly during the experiment. To control convergence, narrow vertical lines of unequal length were drawn across the bands on each target. With the haploscope set for symmetric convergence, the targets were carefully adjusted by the observer so that the dots appeared to lie in the gap of the line. Reliance was placed on the fusion of the two black lines in the periphery to hold the directions of the eyes. If a difference in size was introduced between the ocular images in the vertical meridian, the observer saw the dot displaced vertically from the gap. The magnitude of this displacement could be determined by the distance that the target with the dot had to be moved along the haploscope arm or by using suitable size lenses before the eyes, which would cause the dot to reappear in the center of the gap. In order to test the efficacy of these targets, various size lenses were placed in front of one eye and corresponding displacements of the dot were measured by movements of the target. To offset any vertical fixation disparity and

inequality in the targets, measurements were first obtained with the fusion lines below and then with the fusion lines above the fixation device.[13] The results showed that the observer responded accurately to these targets. Accordingly, they were considered suitable to be used with the eyes in asymmetric convergence. The data obtained by one observer are given in table 4. These and the data of another observer are illustrated in figures 11 and 12. Again, the theoretic change for a complete compensation of the relative sizes of the ocular images is shown in each graph. The measurements with these targets which involve no patent ocular movements clearly indicate a compensative change in the sizes of the ocular images in the vertical meridian when the eyes are turned into asymmetric convergence.

TABLE 4.—*Data (of R. H. D.) Obtained with a Peripheral Fusion Target Pattern on the Haploscope, Showing the Change in the Relative Sizes of the Ocular Images of the Two Eyes in Asymmetric Convergence, in the Vertical Meridian Only*

	Visual Distance = 33 cm.								
	Left					Right			
No Ocular Movement	20°	15°	10°	5°	0°	5°	10°	15°	20°
	A. Fusion Below Fixation								
Average position of setting—mm.	340	340	338	334	331	329	322	318	316
Mean deviation.....	2	1	1	1	1	1	1	1	2
Ratio of Images R:L..	0.960	0.960	0.966	0.998	1.007	1.013	1.035	1.048	1.055
Mean error in ratio...	0.006	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.006
	B. Fusion Above Fixation								
Average position of setting—mm.	353	347	344	339	331	326	322	317	312
Mean deviation.....	0	0	1	2	1	1	0	1	1
Ratio of Images R:L	0.944	0.961	0.969	0.983	1.007	1.022	1.035	1.051	1.068
Mean error in ratio...	0.000	0.000	0.003	0.006	0.003	0.003	0.000	0.003	0.003

It was found difficult to use these target patterns when turned 90 degrees to measure the phenomenon in the horizontal meridian because of the unsteady fixation disparity and probably also because of a tendency to voluntary ocular movements. No consistent data were obtainable.

After-Image Tests.—The importance of after-image tests in connection with these experiments is fairly obvious, although the interpretation of the results may be difficult. Glowing filaments for inducing the after-images were mounted on the targets of the haploscope and properly screened for the two eyes.[14] The target is illustrated schematically in figure 13. Two tubular lamps with single filaments

13. This assumes, of course, that the fixation disparity is not influenced by the position of the fusable patterns.

14. These experiments were performed in the spring of 1936.

were mounted in a horizontal position, one above and the other below the center of each target. These were separated so as to subtend a vertical visual angle of 10 degrees at a visual distance of 33 cm. Suitable

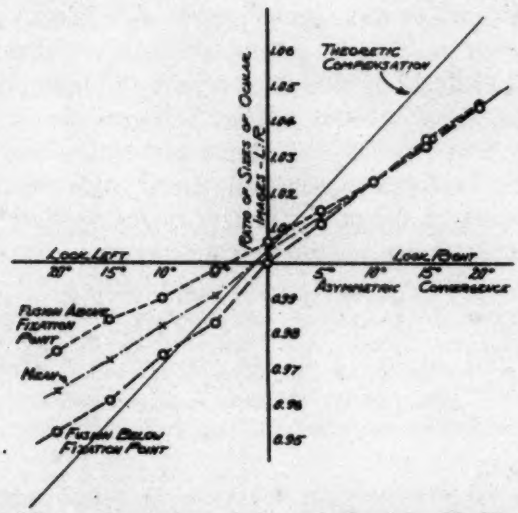

Fig. 11.—Graphic representation of data obtained by K. N. O. on the haploscope with peripheral target patterns at a visual distance of 33 cm.

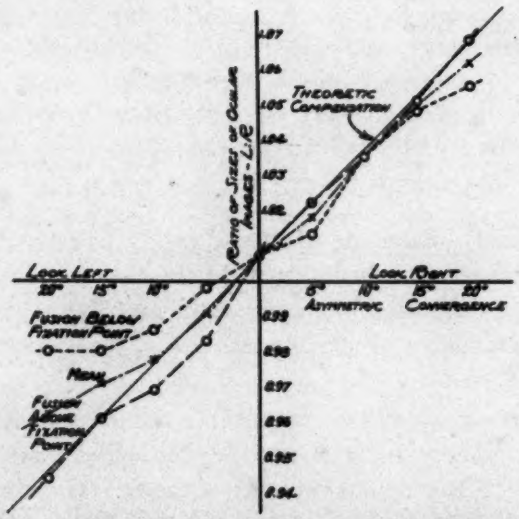

Fig. 12.—Graphic representation of data obtained by R. H. D. on the haploscope with peripheral target patterns at a visual distance of 33 cm.

screens were mounted before the two targets so that each eye saw the opposite halves of corresponding filaments. To hold binocular fixation during the exposure, a suitable target with a horizontal and a vertical

line was set up on the third arm in front of the haploscope so that it could be seen by the observer through half-silvered mirrors. The after-images induced in the eyes by the filaments constituted nonius lines seen in peripheral vision. If the targets were adjusted accurately, an observer without a difference in the sizes of the images saw the after-image lines co-linear. If a difference in size was present, the separation of the lines as seen by one eye was greater than that seen by the other eye.

The experiments were conducted in two ways: First, the apparatus was set for symmetric convergence and the targets adjusted so that the halves of the faintly glowing filaments were opposite. Then the eyes were exposed to the brightly glowing filaments for about ten seconds. The observer then looked at a large white sheet in a position of asymmetric convergence and tried to make a comparison of the separations of the negative half-images. Second, the apparatus was set for an

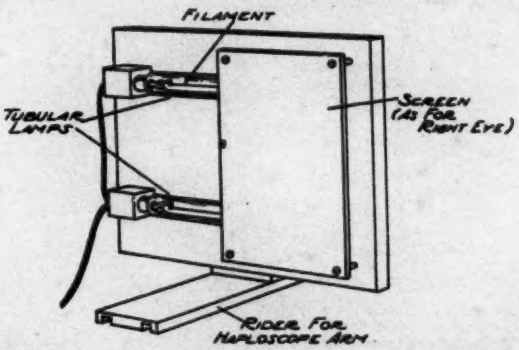

Fig. 13.—Schematic drawing of the targets used on the haploscope for inducing after-images, to study relative sizes of the images when the eyes are turned in asymmetric convergence.

asymmetric convergence position, and with the targets adjusted for equality of image size the eyes were exposed to the bright filaments. The observer then looked toward the large white sheet in symmetric convergence and tried to make a comparison of the separations of the negative after-images of the half-filaments.

Four observers were used in the experiments. At the outset great difficulty was experienced in ascertaining the relative distances between the right and left halves of the after-images, due mostly to the short duration and instability of the images and to the difficulty of attention. Trial and error showed that usually a 4 per cent difference in the sizes of the images could be recognized; therefore, it was believed that the difference due to an asymmetric convergence of 15 to 20 degrees should be discernible.

The experiments, with various modifications, were conducted over a period of several weeks, but on the whole the results were variable

and unreliable. So difficult were the judgments that indications of a trend could not even be ascertained.[15] The results were consistent with the general impression of all observers that data which demanded peripheral judgments of image size from fixed stimuli were usually unsatisfactory and unreliable.[16]

Apparatus with Polarized Pattern.—Obviously, the haploscope apparatus in which the targets always remain the same distance from the two eyes does not duplicate the actual conditions under which the eyes are ordinarily used when turned for an asymmetric convergence. To duplicate actual conditions, a special apparatus was devised which would permit a measurement of the relative sizes of the images in the vertical and horizontal meridians.

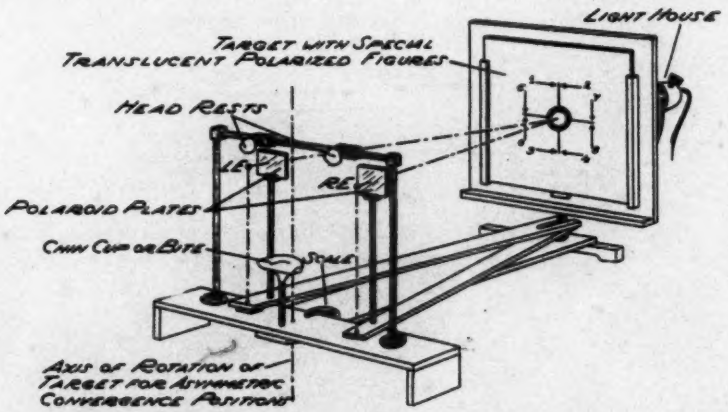

Fig. 14.—Schematic drawing of apparatus using polarized figures for determining the relative sizes of the ocular images of the two eyes when turned in asymmetric convergence.

A negative photographic plate was made of a pattern consisting of lines, arrows and a circle, as shown in figure 14. This was illuminated from behind. Pieces of polarizing film were placed behind the arrows so that the light passing through the even numbered ones was polarized in one plane, while that passing through the odd numbered arrows was polarized in a plane at right angles to that of the even numbered ones. The circle and lines were not polarized. The plate was mounted vertically on an arm which would turn about a vertical axis through a point directly below the midpoint of the centers of rotation of the eyes.

15. These results must be compared, on the other hand, with the after-image tests of W. Herzau and A. Tschermak in which a different technic was used (Herzau and Ogle, cited in footnote 1).

16. This finding is to be differentiated from the nonius-horopter experiments[4] in which kinesthetic factors involved in the movement of the half-images aid in their perception.

The plate then remained perpendicular to the arm. Polaroid plates were also mounted before each of the two eyes with the plane of polarization at right angles to each other but agreeing with those of the arrows. Thus, the observer sees the circle and lines binocularly but can see only the even numbered arrows with one eye and the odd numbered opposing arrows with the other eye.

If the sizes of the ocular images of the two eyes were equal, the opposing arrows would be directly opposite each other, that is, at the same distance from the center of the target, but if the image of one eye was larger, the corresponding arrows seen by that eye would appear

TABLE 5.—*Results Obtained by One Observer (K. N. O.) with the Polarized Target Apparatus**

Degrees of Asymmetric Convergence	Percentage of Correction Necessary to Equalize Images		Geometric Size Difference, Percentage	Apparent Change in Image Size Percentage	
	Vertical	Horizontal		Vertical	Horizontal
	A. Free Fixation (Ocular Movements)				
20° Right.....	0.8 L	1.0 L	7.0 L<R	6.2 R<L	6.0 R<L
15°.....	0.7 L	0.4 L	5.0 L<R	4.3 R<L	4.6 R<L
10°.....	0.1 L	0.5 L	3.3 L<R	3.2 R<L	2.8 R<L
5°.....	0.3 L	0.0	1.6 L<R	1.4 R<L	1.6 R<L
Symmetric.....	0.1 L	0.9 R	0.0	0.1 R<L	0.9 R<L
5° Left.....	0.3 R	1.3 R	1.6 R<L	1.3 L<R	0.3 L<R
10°.....	1.0 R	2.1 R	3.3 R<L	2.3 L<R	1.2 L<R
15°.....	1.5 R	3.5 R 4.0 R	5.0 R<L	3.5 L<R	1.3 L<R
20°.....	1.5 R	2.3 R	7.0 R<L	5.5 L<R	4.3 L<R
	B. Constant Central Fixation				
20° Right.....	5.5 L	5.7 L	7.0 L<R	1.5 R<L	1.3 R<L
15°.....	5.2 L	4.0 L	5.0 L<R	0.2 R<L	1.0 R<L
10°.....	3.0 L	2.3 L	3.3 L<R	0.3 L<R	1.0 R<L
5°.....	2.4 L	2.3 L	1.6 L<R	0.8 L<R	0.7 L<R
Symmetric.....	0.6 R	0.6 R	0.0	0.6 R<L	0.7 R<L
5° Left.....	1.3 R	1.8 R	1.6 R<L	0.3 L<R	0.2 L<R
10°.....	2.9 R	4.1 R	3.3 R<L	0.4 L<R	0.8 R<L
15°.....	4.1 R	5.4 R	5.0 R<L	1.3 L<R	0.4 R<L
20°.....	5.8 R	6.1 R	7.0 R<L	1.2 L<R	0.9 L<R

* The values give the apparent changes in the relative sizes of the ocular images when the eyes are turned in asymmetric convergence. The visual distance was 33 cm. The interpupillary distance was 62 mm.

farther from the center than would the opposing arrows seen by the other eye. By means of lenses of suitable size placed before one eye or the other, the sizes of the images could be equalized and any difference could be measured. If no difference in size could be measured when the target was turned to one side for asymmetric convergence, then a full compensation had occurred between the sizes of the ocular images. Otherwise, a difference in size would have been evident because of the difference in distance of the target from the two eyes.

The data were taken in two ways: first, with free fixation, i. e., with ocular movements, in which case the relative positions of the arrows were observed directly; and second, without ocular movements, in which fixation was maintained at the center of the target and the

arrows were observed in indirect vision. The latter measurements were again found somewhat difficult to make, because of the decreased perceptibility. Table 5 gives the actual data obtained by one observer and the computed relative sizes of the ocular images as obtained by correcting for the geometric difference. Figure 15 shows these data graphically. Inspection of the results again reveals a difference, depending on whether or not ocular movements have occurred in the measurements. With free fixation, nearly a complete compensation is indicated in both the horizontal and the vertical meridian. With constant fixation, on the other hand, the data show only a slight trend in that direction.[17]

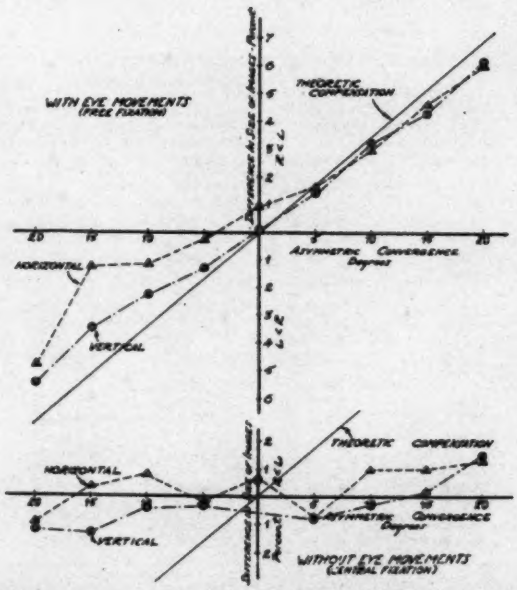

Fig. 15.—Graphic representation of data obtained by K. N. O. with polarized type targets for a visual distance of 33 cm., showing the differences obtained with and without ocular movements.

COMMENT

The results of the experiments described surely indicate that some kind of change in the relative sizes of the ocular images occurs when the eyes are turned in asymmetric convergence. This change compensates remarkably for the difference in the sizes of the retinal images

17. Another way in which the problem can be attacked is by determining the changes in the limits of the regions of single binocular vision. In the vertical meridian this can be done on the haploscope, and in the horizontal meridian it can be accomplished best on the horopter apparatus (see the footnote indicated with an asterisk on the first page). Preliminary data have been obtained by using these methods, but these will not be reported here.

that would naturally result from the difference in the distance of the object from the two eyes. Apart from questions relating to technic, this compensation apparently occurs in the vertical meridian [18] for all persons and in the horizontal meridian for a minority of persons. The compensation increases with the nearness of the fixation point, there being none for distant vision.

The experiments described might be summarized as follows:

Experiment	Result
1. Haploscope	Striking appearance of a compensatory change in the sizes of
A. Circle and square	the images with free fixation, particularly in the vertical meridian; much less change with steady fixation at center, especially in the horizontal meridian
B. Eikonic target	Excellent quantitative results, apparently showing a full compensatory change in the sizes of the images for all visual distances, especially in vertical meridians, and much less in the horizontal meridians for some persons; judgments with peripheral vision not possible because of the small detail of lights and lines
C. Peripheral fusion pattern	A central steady fixation method with peripheral fusion giving excellent results, showing a compensatory change in the sizes of the images in the vertical meridian; not suitable in present design for measurements in the horizontal meridian
D. After-image tests	All tests performed in this laboratory too variable to give reliable data
2. Target with polarized figures	Full compensatory change in the sizes of the images indicated with free fixation, but only slight indication of such change with central fixation and peripheral judgments

From the various experiments with the haploscope in which the angular sizes of the patterns subtended by the two eyes remained the same, a compensatory change in the sizes of the images apparently occurs in the act of the turning of the eyes in an asymmetric convergent position. No signals or innervations for such changes can arise from the patterns themselves. Only in the experiments with the polarized targets could the unequal angular sizes of the patterns give rise to innervations for a compensatory change in the sizes of the images. This fact is of importance in studying the nature of the phenomenon and certainly suggests a myosensory influence of the ocular muscles. It should be pointed out in this connection that equation 1, which

18. In that respect it would logically be of greater importance to compensate for the difference in the sizes of the retinal images in the vertical meridian, since that arising in the horizontal meridian may be reinterpreted through the stereoscopic processes.

expresses the expected geometric ratio (R) of the sizes of the retinal images when the eyes are fixing an object in asymmetric convergence can be written $R = \frac{d_1}{d_2} = \frac{\cos \phi_2}{\cos \phi_1}$ in which ϕ_1 and ϕ_2 are the angles of turning the left and right eyes, respectively, from the primary position to the object in asymmetric convergence (fig. 1). This further suggests that a relation may exist between the apparent sizes of the ocular images, as measured, and the positions of the eyes themselves.

A problem does exist because of the different results found in some of the experiments which do not involve ocular movements. While no adequate explanation for these different results can yet be given, it should be mentioned that in nearly all experiments of this type in which the apparent directions of contours are judged in peripheral vision the results have not been entirely satisfactory.

It has been suggested that the results showing an apparent compensation in the sizes of the images in the experiments in which free fixation was permitted could be due to differential ocular movements and to the failure of the fusible portions of the targets to control the directions of the eyes. This suggestion would seem to be nullified by the experiment with the peripheral fusion target. It would seem highly improbable, moreover, that the magnitude of differential ocular movements in the experiments with the haploscope would be such as to yield the highly consistent data which parallel an image compensation process for all visual distances.

What causal factors could be responsible for this phenomenon cannot, at present, be stated with any degree of assurance. One possible explanation may be an actual change in the relative sizes of the retinal images due to changes in the dioptric systems of the eyes. It is believed, however, that the phenomenon could not be due to a differential accommodation of the two eyes. The changes, then, if within the dioptric systems would have to be of a more complicated nature.

Another explanation might be that an influence has been brought to bear on the retinal membrane itself; for example, through the changed tension conditions of the muscles of the eyes in asymmetric convergence, which would arise, possibly, from an indirect myosensory influence or a direct mechanical action. The relative apparent sizes of the images in asymmetric convergence seem to be closely related to the relative positions of the two eyes in the head.

A third possible explanation might be a psychologic reinterpretation within the higher cortical regions of the brain but dependent again on some sensory influence of the ocular systems when the eyes are turned in asymmetric convergence. It was hoped that after-image experiments would aid positively in differentiating between these possibilities.

It will not be the purpose of this paper to attempt an exhaustive consideration of these possibilities but merely to present additional evidence which tends to establish some type of compensatory process within the ocular systems, not, however, without indicating some of the problems involved in its measurement.

SUMMARY

The problem of the relative sizes of the ocular images with the eyes in asymmetric convergence was considered further by using other methods of experimentation. In general, however, the results were a confirmation of those described previously by Herzau and Ogle, which indicated that some type of change in the relative sizes of the images occurs which offsets or tends to offset the difference in distance from the object to the eyes in asymmetric convergence. This apparent change was in the direction and of the amount necessary to compensate for the difference in the sizes of the retinal images that would exist because of the difference in the distance of an object from the two eyes for all visual distances from 5 meters to 20 cm. No change could be measured with the eyes adjusted for distant vision. The compensatory change was found principally in the vertical meridian, though it occurred for some observers in the horizontal meridian. The phenomenon apparently takes place in the act of the turning of the eyes to an asymmetric convergence position. The results also differed, depending on whether ocular movements were involved in the measurements, those obtained by judgments of contours peripherally seen showing much less change in the sizes of the images. After-image tests proved unsatisfactory. No attempt was made to determine the causal factors which could be responsible for such a phenomenon, though three possibilities were suggested: an actual change in the sizes of the retinal images from dioptric changes, a change brought to bear on the retinal membrane and a psychologic reinterpretation, all dependent on some sensory influence of the eyes themselves when turned in asymmetric convergence.[19]

Various members of the staff assisted in obtaining data.

19. Since the preparation of this manuscript a paper has appeared (Schubert, G.: Zur "Aniseikonia"-Frage, Arch. f. Ophth. **140**:55, 1939) in which an attempt is made to explain this phenomenon by a special involvement of the receptor and motor apparatus of the two eyes. On the basis of the argument presented in that brief paper, the interpretation seems inadequate. Certainly in view of the large amount of data obtained with different experimental arrangements by a number of observers, that explanation requires further elucidation and experimental confirmation.

Clinical Notes

EVALUATION OF THREE METHODS COMMONLY USED IN EXAMINATION OF EYES OF SCHOOL CHILDREN

BERNICE C. ENGLISH, HARRISBURG, PA.; B. CECELIA SHMUKLER, M.D., PITTSBURGH, AND ALFRED COWAN, M.D., PHILADELPHIA

This study was undertaken for the purpose of determining the relative value of each of the following methods: the visual safety telebinocular method (Betts), the N.E.A.M.A.[1] method of testing vision and the ordinary routine medical inspection in schools. For the purpose of this investigation all children tested were taken from the third grade. Their ages ranged between 8 and 10 years. This was done in order to eliminate as far as possible any variable factors which might be influenced by age, experience or intelligence. The N.E.A.M.A. method and the Betts method were carried out by the same school nurses. Four hundred and eighty-five children were tested by all three methods. A description of these three screening methods is presented.

SCHOOL MEDICAL INSPECTION

The manual issued by the Pennsylvania State Department of Health presents the following instruction for the school medical inspector:

> Hang the Snellen chart against a dark background and with a good side illumination, the chart approximately on a level with the pupil's eyes when taking the test. . . . Have the pupil seated at a measured distance of twenty feet from the chart. Each eye must be examined separately. After testing the visual acuity, the lids, conjunctiva, cornea and eyeballs should be examined for abnormalities.

N.E.A.M.A. METHOD

Apparatus Required.—The letter chart (Snellen scale) is commonly used for the N.E.A.M.A. method. It is an advantage to obtain a chart not easily memorized. For young children the symbol E chart (Snellen scale) is used, and the child points the direction in which the prongs of the E face, the chart being placed at a distance of 20 feet (6 meters).

From the State Council for the Blind, Department of Welfare, Commonwealth of Pennsylvania.

This study was conducted cooperatively with the Department of Public Instruction and the superintendent of schools of Erie County.

1. Conserving the Sight of School Children: Report of the Joint Committee on Health Problems in Education of the National Education Association and the American Medical Association with the Co-Operation of the National Society for the Prevention of Blindness, Publication 6, Washington, D. C., National Education Association, 1929.

Technic of Testing.—Position and Lighting of Chart: The chart should be hung with the 20 foot line approximately on a level with the child's eyes. Some children are restless unless seated.

Light should be sufficient and without glare. There should be not less than 10 foot candles evenly distributed over the chart (a foot candle meter is of great assistance in determining amount of illumination). Whenever possible, artificial light should be used in order to keep the illumination constant at all times, on cloudy as well as on bright days.

Distance: Examinations should be made in a room the length of which is at least a little more than 20 feet. A distance of 20 feet from the chart should be accurately measured and marked.

Position of Child: The child should be placed in a good light, but so that the light does not shine from in front or from the side directly into his eyes; it must shine directly on the chart, however, without glare.

The child should stand or should be seated on a chair on the 20 foot line, so that his eye is exactly 20 feet from the chart.

Reading by the Child: If the child wears glasses, they should be removed and a test made first without glasses, followed by a test with glasses. If only one test is recorded, it should be the one with glasses.

A small card (about 3 by 5 inches [8 by 13 cm.]) or a piece of heavy paper folded to 3 by 2½ inches (8 by 6 cm.) is held before one eye of the child, the card or paper being placed obliquely against the nose so as to cover the eye completely and avoid any pressure on the eyeball. Both eyes are to be kept open. One eye is tested at a time. It is wise to standardize technic by always beginning with the right eye.

The child is asked to read with the uncovered eye. If the examiner has an assistant, the examiner points to the letters or characters on the chart, being careful that there is no memorization of the letters and being sure that the child is encouraged to do his best reading. If an assistant is not available, the examiner will have to approximate this technic as seems wisest under the circumstances, deciding whether he shall stand near the child or near the chart.

One should begin the test at one of the lines of large symbols or letters and run down rapidly to the child's easy limit, thence carefully seeking identification of each character by pointing if the symbol E is used or by naming the letter if letter charts are used.

Standards.—More than two errors to a line indicates inability to read that line. Measurements are recorded by the use of figures such as 20/20, 20/30, 20/40, etc., the first number being the distance from the chart and the second number the number of feet at which the normal eye would read the line read (the number on the chart at the line read).

In addition to testing the child's visual acuity, it is equally important to determine whether there are any signs or symptoms which suggest ocular disturbances. Such signs and symptoms will be associated with defects of visual acuity and diseases of the eye.

Signs and symptoms associated with disturbances of visual acuity are: (*a*) an unsatisfactory test with the symbol or letter chart; (*b*) the holding of the head in a peculiar position while reading the chart, as in some cases of astigmatism; (*c*) a teacher's report that although a child can read 20/20 at the examination he cannot apply himself to study for a long time without suffering definite or indefinite discom-

forts, as from headaches and sore or watery eyes; (*d*) a history of headaches and sore eyes associated or not with ability to see distant objects better than objects held within a few inches of the eye, and (*e*) a history of blurring vision, confusion in recognizing words and loss of the line in reading.

Signs and symptoms associated with diseases of the eye or its appendages are, in general: (*a*) any redness or swelling of the eyeball, the inside of the eyelids or the edges of the eyelids and any scaling of the edges of the lids or loss of lashes; (*b*) any discharge from the regions of the eye, especially from the inner corner of the opening between the two eyelids (may be water, mucus or pus); (*c*) any little lumps inside the eyelids, as in granular eyelids (chalazion); (*d*) any whitish or grayish areas on or apparently in the eyeball, and (*e*) any variation of the black spot (pupil) at the center of the colored part of the eyeball, i. e., a tiny pupil (pinpoint, as with the use of certain drugs like morphine) or a large pupil (dilated, as in darkness or with the use of certain drugs like atropine which are used in some medicines for the eyes). The pupils should be the same size in the two eyes, should be circular and should react equally to light.

BETTS METHOD

The Betts ready to read tests, which employ the visual safety telebinocular, are as follows.

Test 1—an introductory test to demonstrate binocular vision
Test 2—a test for distance fusion
Test 3—a test for visual efficiency
- (*a*) Two eyes
- (*b*) Left eye
- (*c*) Right eye

Test 4—a test for vertical imbalance
Test 5—a test for coordination level
Test 6—a test for lateral imbalance
- (*a*) Distance
- (*b*) Reading distance

Test 7—a test for reading distance fusion
Test 8—a test for sharpness of image
- (*a*) Reading distance
- (*b*) Distance

OPHTHALMOLOGIC EXAMINATION

The ophthalmologic examination in this study included precycloplegic, cycloplegic and postcycloplegic tests. The precycloplegic examination consisted of the determination of the visual acuity with and without correction, inspection of the external appearance, ophthalmoscopic examination, study of the muscle balance for far and near vision, duction tests and tests for accommodation and stereopsis. One drop of a 1 per cent solution of atropine sulfate three times a day was instilled ten times in every case, the last drop having been instilled on the morning of the examination for refraction.

The following form was filled out for each child:

STATE COUNCIL FOR THE BLIND
Department of Welfare
Harrisburg

Name Age
Address
School Grade
History: General
Ocular
Symptoms
Precycloplegic Examination Date
Visual acuity:
Without correction: O. D. O. S. 02
With correction: O. D. O. S. 02
External examination:
Ophthalmoscopic examination:
Muscles
Distance (6 meters)
Near (33 cm.)
Ductions: Prism convergence
Prism divergence
Near point of convergence
Accommodation
Extraocular movements
Stereopsis
Cycloplegic Examination Cycloplegic used:
Date:
Ophthalmoscopic examination:
Static refraction: Trial case:
O. D. O. D.
O. S. O. S.
Postcycloplegic Examination Date:
Trial case:
O. D.
O. S.
Muscles
Distance (6 meters)
Near (33 cm.)
Ductions: Prism convergence
Prism divergence
Near point of convergence
Accommodation
Extraocular movements
Stereopsis

Ophthalmologist .....

A child was considered as having failed to pass the preliminary examinations unless he reacted normally to all tests by any one of the three methods of screening.

It was found that of the 485 children examined, 301 failed to pass one or more of the preliminary examinations. Of the total (485), 111 were given ophthalmologic examinations. This number included 91 children who had failed to pass one or more of the preliminary examinations and 20 who had passed all three methods of examination. The 20 children who were found by all three methods to have practically normal eyes from every standpoint were used as a check and for whatever information might be revealed. It was deemed advisable that the ophthalmologist should have no knowledge of any of the previous findings, so that the examinations could be conducted without prejudice. The results of the preliminary examinations follow:

Number of children who failed to pass medical inspection	77
Number of children who failed to pass the N. E. A. M. A. method	124
Number of children who failed to pass the Betts method	260
Number of children who failed to pass one or more examinations	301
Number of children who failed to pass all three examinations	40
Number of children who passed all examinations	184
Total number of children examined by all three methods	485

ANALYSIS OF THE RESULTS OF THE OPHTHALMOLOGIC EXAMINATIONS OF 111 CHILDREN

The group of 20 children used as a check and who were found to have practically normal eyes by all three methods were eliminated after examination by the ophthalmologist. It can be said, therefore, that if all three methods are applied, those children eliminated can be considered as having no visual disability

The proportion of children in the remaining group of 91 who were suspected of having some indication of ocular defect and who were referred to the ophthalmologist were detected by each method as follows:

By medical inspection	48.2 per cent
By the N. E. A. M. A. method	47.2 per cent
By the Betts method	86.8 per cent

The school medical inspector referred 38 children for ophthalmologic examination, 25 of whom were found to be in need of correction. There were 53 children passed by him as having no need of attention, 17 of whom were found to have materially defective eyes. By this method, therefore, 17 children with an ocular defect were missed and 13 were needlessly referred for examination.

By the N.E.A.M.A. method 43 children were sent to the ophthalmologist, 35 of whom were found to have ocular defects. By this method 48 children were passed as having no indication of an ocular defect, and of the 48, 7 were found by the ophthalmologist to have ocular defects. Thus, by the N.E.A.M.A. method 7 children were missed and 8 were needlessly referred.

The nurses using the Betts method referred 79 children to the ophthalmologist, of whom 35 were found to be in need of correction. Twelve children were passed by this method as having no defects, yet 7 were found to be in need of correction. Forty-four children were needlessly referred.

The results of three methods of examination according to complete findings of the ophthalmologist appear in the accompanying table.

Results of Ophthalmologists' Findings with Three Methods of Examination

Method	Percentage of Children Properly Referred by Each Method and Found by Ophthalmologist to Be in Need of Correction	Percentage of Children Passed by Each Method but Found by the Ophthalmologist to Be in Need of Correction	Percentage Referred Needlessly
School medical inspection	59.5	22.0	34.2
N. E. A. M. A.	83.3	14.5	18.6
Betts method	83.3	58.3	55.6

In conclusion, it might be stated that the figures in the table speak for themselves. The Betts method will appear to be 83.3 per cent correct in referring children for ophthalmologic examination; but when it is considered that 58.3 per cent were missed by the Betts method as compared with 14.5 per cent missed by the N.E.A.M.A. method and that 55.6 per cent were referred for examination needlessly on the basis of the Betts method as compared with 18.6 per cent referred needlessly on the basis of the N.E.A.M.A. method, it need hardly be stated that the latter method is superior to the former for the purpose of these examinations. Furthermore, it seems from a careful analysis of the specific findings in the various cases that the N.E.A.M.A. method is better from a medical standpoint than the Betts method For example, of the 7 children missed by the Betts method, 1 was missed by the N.E.A.M.A. method. Four of these were picked up by the N.E.A.M.A. method on the basis of symptoms, and 2 were picked up because of failure to pass the test for visual acuity. The ophthalmologist found that the 4 children missed by the Betts method but picked up by the N.E.A.M.A. method because of symptoms had a correctible amount of hypermetropia, and the remaining 3 missed by Betts but picked up by the N.E.A.M A. method because of faulty visual acuity had a correctible amount of myopia.

A good example of the practical efficiency of the N.E.A.M.A. method over the Betts method is shown by the fact that the children missed by the N.E.A.M.A. method had moderate amounts of hypermetropia, which, because of lack of ocular and behavior symptoms, were well and easily tolerated; among the 7 children missed by the Betts method, 3 were myopic, which indicates that the test is faulty even in regard to visual acuity. Faulty visual acuity could hardly and did not in any instance pass the N.E.A.M.A. test. This would indicate, then, that unless there are symptoms present the N.E.A.M.A. method may fail to detect hypermetropia, while the Betts method may fail to detect either myopia or hypermetropia.

The Betts method is probably a more useful and a more thorough means of examination from a psychologic standpoint, but for the purpose of these examinations the medical rather than the psychologic standpoint is preferred.

This investigation would also seem to prove that the ordinary method of routine medical inspection to determine visual defects in school children is unreliable.

AFTER-CATARACT

ALFRED COWAN, M.D., AND ROBB MCDONALD, M.D., PHILADELPHIA

Despite the fact that the extraction of the cataractous lens is probably the most frequently performed ophthalmic operation, there have been surprisingly few reports of the anatomic changes after operation. Within the past few years Knapp,[1] Ellett,[2] Greenwood and Grossman,[3] O'Brien[4] and others have reported before the American Ophthalmological Society the visual results and immediate complications of cataract operations. One of us (A. C.[5]) a number of years ago reported on the anterior limiting membrane of the vitreous. However, we feel that the subject is still of sufficient interest to warrant this presentation.

This report is based on the biomicroscopic appearance of 218 eyes one or more years after extraction of senile cataract. The purpose of the investigation was merely to note the late anatomic or pathologic changes in an aphakic eye. No attempt has been made to compare the visual results or to evaluate the hazards of one method of extraction against those of another. All patients were from the clinics of the Wills Hospital. The records from 1930 to 1937 were searched for reports of operations on senile cataract, and only those persons who had no pathologic changes other than a senile cataractous lens which had been removed were asked to come to the hospital. Approximately 1,000 cards were sent to patients living in Philadelphia or suburbs asking them to return for an examination. For various reasons comparatively few responded, the total number of eyes examined being 218. The following operations had been done on this group of patients: simple extracapsular extraction, 16; simple intracapsular extraction, 1; combined extracapsular extraction, 127, and combined intracapsular extraction, 74. The interval of time between the operation and the examination varied from one to seven years, the average being three and thirteen hundredths years. The operations were performed by the various members of the surgical staff of the hospital, and the series as a whole consisted of patients who at the time of their last visit to the clinic were considered to have had a successful operation, since no person whose end result was considered a failure was asked to report.

From the Laboratory of Ophthalmology, the Wills Hospital.

Read at the Seventy-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 5, 1939.

1. Knapp, A.: The Complications of the Forceps in Intracapsular Cataract Operation, Tr. Am. Ophth. Soc. **34**:162, 1936.

2. Ellett, E. C.: The Results of Cataract Extraction Five and More Years After Operation, Tr. Am. Ophth. Soc. **33**:341, 1935.

3. Greenwood, A., and Grossman, H. P.: An Analysis of 1,343 Intracapsular Cataract Extractions, Tr. Am. Ophth. Soc. **33**:353, 1935.

4. O'Brien, C. S.: Detachment of the Choroid After Cataract Extraction, Tr. Am. Ophth. Soc. **33**:325, 1935.

5. Cowan, A.: Concerning a Membrane Between the Vitreous and Anterior Chamber Seen After Removal of the Crystalline Lens and Its Capsule, Tr. Am. Ophth. Soc. **29**:179, 1931.

The findings, therefore, represent a good cross section from the various clinics, the operations having been performed by a group of men of average ability, including the skilful as well as the not so skilful surgeon.

RESULTS OF OBSERVATIONS

Some inflammation probably takes place in any eye on which an intraocular operation is performed, and direct evidence, as shown by precipitates on the posterior surface of the cornea, ruptures and folds in Descemet's membrane, endothelial dystrophy, cells in the aqueous and organized exudate from the iris or vitreous, was found in 82 instances (37.6 per cent) regardless of the method of extraction.

Fig. 1.—The anterior hyaloid membrane after intracapsular extraction: *1*, intact hyaloid membrane after successful intracapsular extraction; *2*, hernia with intact hyaloid membrane; *3*, rupture of the hyaloid membrane with prolapse of the vitreous; *4*, almost perfect hemispheric hernia (seen in 4 instances); *5*, parallel folds in a loose but intact hyaloid membrane, and *6*, hyaloid membrane drawn forward and incarcerated in the corneal scar.

Obviously, all these patients can be considered to have recovered more or less. We have no record of the eyes lost because of postoperative inflammation, as we were concerned only with the patients who were discharged from the clinic as cured. Active inflammation was found, however, in 3 eyes on which the combined extracapsular extraction was done; they all had less than 6/60 vision when refraction was done at the time of biomicroscopic examination.

In every eye there was some loss of transparency along the scar of the incision; fairly widespread grayish infiltration and scarring, superficial or deep, not only in the vicinity of the incision but in other parts of the cornea, were found in 44 eyes (20 per cent).

Disturbance of the iris pigment had taken place, almost without exception, in every eye. It is true that a certain amount of migration and absorption of pigment can be considered normal in senile eyes, but there is no doubt that the changes are greater after any intraocular operation. Pigment granules on the posterior surface of the cornea were seen in nearly every eye. There is always atrophy of both the stroma and the retinal pigment layer along the pillars of the coloboma after iridectomy. Wide sheets of the retinal pigment layer were often either entirely absent or detached and carried away from their normal

Fig. 2.—Anterior capsule incarcerated in the corneal scar and a large clump of Hirschberg-Elschnig bodies in the anterior chamber after extracapsular extraction. Vision was 6/9—.

situation. Sometimes they lay on the surface of the iris and sometimes in the coloboma. Whenever the iris is cut or traumatized in any way, some atrophy of the iris immediately surrounding the injured portion takes place. After allowances were made for what might be ordinarily expected, abnormal widespread disturbance of pigment was found in 58 eyes (26.6 per cent), and extensive, well defined areas of general or or more or less advanced atrophy of the iris were found in 82 (37.6 per cent) eyes. Strangely, in 16 of the 17 eyes in which the cataract was extracted without iridectomy the iris was considerably atrophied in a wide tract extending from the upper margin of the pupil to the limbus above. In these eyes the pupil was always vertically or obliquely

oval, pear shaped or slitlike. The 1 person who had undergone a simple intracapsular operation presented an almost perfectly round central pupil with an intact iris.

Anterior synechia along the limbic scar is so common that it may be considered, at least to some extent, as the natural sequence of iridectomy. Either one or both roots of the pillars of the coloboma are usually incarcerated in the scar, whether the iridectomy was done before or at the time of the extraction. Also, with or without iridectomy, the iris is often adherent to the cornea over a part or along the entire limbic scar outside of the coloboma. The number of anterior synechiae, small and extensive, was 163 (74.7 per cent).

Considerable organized exudate or connective tissue, adherent to the upper part of the cornea without other evidence of marked inflammation, was found in 18 eyes. Accessory colobomas caused by misdirection of the knife were seen in 4 eyes; in 3 they were caused by false counterpuncture and in 1 by both the initial puncture and counterpuncture. Some form of unnecessary mutilation of the iris, resulting in a ragged, misshapen or incomplete coloboma or iridodialysis, was found in 40 eyes. Prolapse of the iris was present in 12 instances.

Discission after an extracapsular extraction was usually but not always found to involve the hyaloid membrane. Frequently the anterior lens capsule, the hyaloid membrane or the vitreous were drawn into the wound. Prolapse of the vitreous into the anterior chamber was found in 58 instances. It occurred in 18 of the 75 eyes that were operated on by the intracapsular method. It is a remarkable fact that the normal vitreous never mixes with the aqueous whether the hyaloid membrane is intact or not. Another peculiarity which has never been explained is the strange affinity uveal pigment has for the vitreous. Either the hyaloid membrane or the vitreous always seems to contain a great number of brown pigment granules.

Varying degrees of hernia of the anterior hyaloid membrane were seen in 25 eyes. A most peculiar type of hemispheric form was found in 4 eyes. A complete pupillary membrane, consisting of both an anterior and a posterior capsule, was seen in 15 eyes. In 2 eyes on which intracapsular extraction was done, the hyaloid membrane was dense and translucent, and the patients had vision of only 6/12 + 3 and 6/15. In 4 of the eyes for which intracapsular operation was recorded, considerable capsular remains indicated failure to deliver the lens complete in capsule.

Globular transparent bodies, Hirschberg-Elschnig [6] bodies, are common, having been seen 54 times (24.7 per cent). That they are either enlarged capsular epithelial cells or proliferations of the capsular epithelium seems to be proved by the fact that they are never seen in the absence of an anterior lens capsule. They were present only in the eyes on which extracapsular extraction was done and were attached to the remaining anterior lens capsule. They interfere with vision only when they fill the pupil but are liable to grow for years. Small whitish granular bodies, often described as resembling insect eggs, were seen in 5 eyes. These also are probably the result of some process which takes place in the anterior lens capsule.

6. Cowan, A., and Fry, W. E.: Secondary Cataract, Arch. Ophth. **18**:12 (July) 1937.

Posterior synechia is another common finding. It occurred nearly always to some extent after extracapsular extraction and in 51 eyes after intracapsular extractions.

Threads of cotton were seen twice and an eyelash once in the anterior chamber, apparently without causing any irritation. An incomplete Sommering ring occurred in 2 eyes.

CONCLUSIONS

After extraction of senile cataract almost every aphakic eye shows evidence of having been subjected to tremendous insult, regardless of the skill of the operator or the method used. The successfully performed intracapsular extraction results in a better eye, because of the complete removal of the capsule with the lens substance. From the standpoint of useful vision, capsular remains do not in themselves interfere as long as there is an adequate opening. On the whole, the visual acuity of the group of patients subjected to extracapsular extraction was almost as good as that of the patients on whom intracapsular extraction was done; but without lens capsule there are not only fewer immediate sequelae but less likelihood of further complications arising from the retained capsule. Rupture of the hyaloid membrane, prolapse of the vitreous, incarceration of the capsule or vitreous in the corneal wound, atrophy of the iris and irregular, mutilated or displaced pupils do not in themselves interfere with good vision. Good vision does not always follow the best operative result. A number of patients who could be considered to have as nearly perfect an operative result as possible and who did not show any objective signs of pathologic changes elsewhere in the eye obtained vision of only 6/15 to 6/9 with the best correcting lens.

There seems to be no justification as far as usefulness or even the appearance of the eye is concerned for doing a simple extraction. As we have stated, in nearly every eye in which the extraction was done without iridectomy the iris showed marked atrophy with a displaced and irregular pupil. It is a mistake to believe that complete iridectomy destroys the pupillary reaction. If it is free, the movements of the intact remaining border of the pupil seem perfectly normal.

IMPORTANCE OF THE ANGLE DELTA IN LOCALIZING INTRAOCULAR FOREIGN BODIES

Report of Illustrative Case

M. E. ALVARO, M.D., SÃO PAULO, BRAZIL

The optic axis of the globe only exceptionally coincides with the visual axis. The angle formed by these two axes has had several denominations, according to the points of reference. Thus the angle formed by these two axes when their intersection is at the nodal point of the eye had been called angle alpha by Helmholtz.[1] The angle formed

1. Helmholtz, H.: Helmholtz's Treatise on Physiologic Optics, translated from the third German edition by J. P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1924.

by the line uniting the rotation center of the eye to the point of fixation with the optic axis is the angle gamma (Donders [2]). The angle formed by the central pupillary line, that is, a line perpendicular to the cornea and passing through the center of the pupil and the visual axis, is the angle kappa (Landolt [3]). Helmholtz' angle alpha was called beta by Brubaker,[4] and Landolt's angle kappa was christened delta by Howe.[5] In 1866 Helmholtz [6] himself gave a definition of angle alpha, according to which it was formed by the intersection of the visual axis and the major axis of the corneal ellipse. Later it was found that the cornea is not a true ellipse, having a more or less irregular form, so that Helmholtz' definition became meaningless. The angle kappa, apparently the easiest to be measured, is only really equivalent to that angle, according to Landolt's [7] definition, when measurements are made which take into consideration the reflex of a light on the cornea at a part corresponding to the center of the pupil, and not all methods used to measure that angle do that. A few repair only the geometric center of the cornea.

It is true, however, that the angle alpha, or its synonym, the angle beta, the angle gamma and the angle kappa, with its synonym, the angle delta, have nearly the same value, at least for clinical purposes, if their respective measurement is taken at a distance of more than 30 cm. (fig. 1).

Generally, the angle that is measured is angle kappa, as most methods use the reflected image of a light on the cornea. There are several instruments for measuring these angles, of which I shall mention the perimeter, the synoptophores, Duke-Elder's,[8] Dashevsky and Booshmitch's,[9] Howe's [10] and Comberg's, and Alvaro's [11] instrument.

With the perimetric method, the patient is made to look at the center of the apparatus while a light is moved along the arc until the reflex of that light appears to be in the geometric center of the cornea. Direct reading on the instrument gives one the value of the angle kappa it

2. Donders: On the Anomalies of Accommodation and Refraction of the Eye, translated by W. D. Moore, London, New Sydenham Society, 1864.

3. Landolt, E., and Landolt, M.: Defective Ocular Movements and Their Diagnosis, translated by A. Roemmele and E. W. Brewerton, London, Oxford University Press, 1914.

4. Brubaker, in Duke-Elder,[8] p. 759.

5. Howe, cited by Cords, R., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 3, p. 468.

6. Helmholtz, in Duke-Elder,[8] p. 759.

7. Landolt, in Duke-Elder.[8]

8. Duke-Elder, W. S.: Textbook of Ophthalmology, London, Henry Kimpton, 1932, vol. 1, p. 760.

9. Dashevsky, A. I., and Booshmitch, D. G.: Optical Decentration of the Eye, Am. J. Ophth. **21**:125 (Feb.) 1938.

10. Howe,[5] p. 740.

11. Alvaro, M. E.: Apparelhos para medição do estrabismo, in Actas Cong. argent. de oftal., Buenos Aires, 1937, vol. 1, p. 392.

being positive when measured at the homonymous half of the perimeter, as seen by the patient, and in its relation to the examined eye, and negative when measured at the opposite half. The method employing the synoptophores is somewhat similar in its principle.

With Duke-Elder's method, the patient is told to look at a light at the center of the perimeter, and the examiner tries to find a spot along the arc where the reflex of that light coincides with the center of the patient's pupil. As the angle of incidence is equal to the angle of reflection, half of the number of degrees found at the perimeter will be the equivalent of the angle kappa.

Dashevsky and Booshmitch have the patient look at the center of the perimeter and move a light along the arc; they observe its reflex from behind it and try to locate the point of the extreme limits of the cornea where the reflex disappears due to the change in radius. Two different values are generally found for each half of the cornea, and the angle kappa will be half of the difference found. My instrument, shown at the scientific exhibit of the Fifteenth International Ophthalmologic

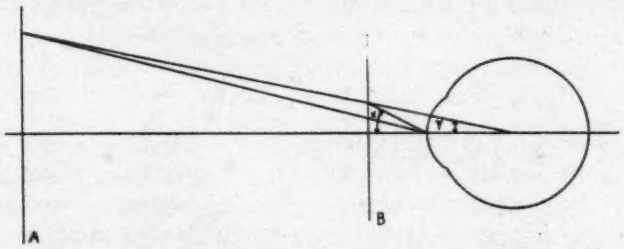

Fig. 1.—Measured at *A*, angle kappa is only slightly larger than angle gamma. Measured at *B*, angle kappa is much larger than angle gamma.

Congress in Cairo, is based on the same principle, that is, an attempt to locate the geometric center of the cornea. With this instrument, by means of two beams of light that are made to fall simultaneously at two diametrically opposed points of the sclerocorneal limbus, the geometric center of the cornea is located by a light moving along an arc rigidly connected with the sclerocorneal lights, at which the patient is made to look; when the two reflexes reach the limbus, the angle kappa may be read on the arc at the point where the movable light is found.

With Howe's method, an ophthalmometer after Javal or Techerning's ophthalmophacometer is utilized, and the relative position of the corneal and lens reflexes are compared. It is a rather slow procedure but accurate.

It is common knowledge that the visual axis crosses the cornea eccentrically, not only in relation to the horizontal diameter but in relation to the vertical one, and the point of intersection of that axis with the surface of the cornea is generally above and nasal to the center of the same; or, in other words, when the eye gazes at an object, the anterior

pole of the globe is directed below and to the outside (temporal) of the object. But although this fact has been known for many years, only recently was this vertical deviation actually measured in a large series of cases, and credit is due Dashevsky and Booshmitch for the accurate measurement of the combined vertical and horizontal deviation of the optic axis from the visual axis. For the newly devised vertical angle, these investigators suggested provisionally the name of angle delta, calling the angle measuring the combined vertical and horizontal deviation angle kappa-delta.

All ophthalmologists know how important it is to measure angle kappa (alpha-gamma-kappa) when one wants to make accurate measurements, which are essential for correction of squint and for the location of intraocular foreign bodies and retinal tears. When that angle is of a small value, it may be overlooked, because the surgical methods have not so far reached the accuracy of fractions of a millimeter, but when the angle kappa (alpha-gamma-kappa) is large and is not taken into consideration, neglect of its measurement may cause great damage to the patient. The case I shall report is a good illustration of this fact and points out especially the importance of measuring the vertical deviation too.

REPORT OF CASE

W. K., a German aged 29, a resident of a small town in São Paulo, Brazil, was examined on May 24, 1938. He stated that he had suffered trauma to his left eye four months before and that he was under the impression that "something had flown into his eye." The physician (not an ophthalmologist) who had treated him thus far had told him that nothing resembling a foreign body could be seen. His sight, however, began to decrease, and the eye was inflamed and tender. When I examined him his vision was 1/100. There was pronounced ciliary injection. Few cells could be seen in the aqueous, there was slight siderosis of the crystalline lens and the pupil was dilated. Few pigment deposits were seen on the anterior crystalline lens, and there were many cells in the vitreous, which rendered retinoscopic examination impossible. A small foreign body could be seen in the cornea; it was removed, but obviously it could not have been the cause of the ocular inflammation. No signs of a foreign body could be traced either in the cornea or in the iris or lens, nor were there any indications of the passage of one through these several layers of the eye. A roentgenogram was requested, and 10 cc. of atophanyl [12] was given intravenously every day. The roentgen examination showed an intraocular foreign body, and with Comberg's [13] method it was possible to find the position of the body, just 1 mm. below the optic disk.

With injections of atophanyl the vitreous cleared up rapidly, and one week after the first examination of the patient examination of the fundus enabled one

12. This preparation is a combination of cinchophen sodium (0.5 Gm.), sodium salicylate (0.5 Gm.) and procaine hydrochloride (0.008 Gm.) in 5 cc. of solvent.

13. Comberg, W.: Ein neues Verfahren zur Röntgenlokalisation am Augapfel, Arch. f. Ophth. **118**:175, 1927.

to see a foreign body in the retina above the optic nerve. The patient suspected the foreign body to be a piece of steel or iron, and an attempt was made to remove it from its posterior position with the aid of a giant magnet. After three successive trials it was finally possible to see the foreign body moving freely in the vitreous, and subsequently, through a small incision of the sclera and with the help of Hirschberg's small magnet, it was successfully removed. Within three weeks after the operation the patient's sight increased to 2/3, and he was discharged.

There could not have been any question of the roentgenologist's ability in locating the foreign body, and I knew that he had taken the patient's angle kappa into consideration, but as the error had been in the vertical plane and as I had read the results of Dashevsky and Booshmitch's survey, I decided to check on the vertical deviation, or angle delta. Using the method of these authors, I was able to find that the patient had a negative angle delta of 15 degrees, or, in other

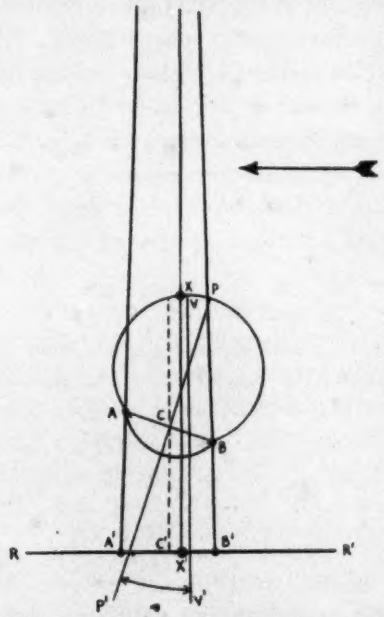

Fig. 2.—The position of the patient's body is indicated by an arrow. The size of angle delta is exaggerated to render the picture more easily understood. Looking at *V'*, the patient's optic axis was at *P-P'*. The foreign body at *X*, really above the posterior pole of the globe, *P*, showed at plate *R-R'* below point *C'*, corresponding to point *C*, which is equidistant of *A* and *B*, the two opaque points in the contact glass, and so induced to error.

words, that the visual axis crossed the cornea 15 degrees below its center. This vertical deviation explains satisfactorily why with Comberg's method the foreign body appeared to be 1 mm. below the optic nerve when it really was 1 mm. above it (fig. 2).

COMMENT

In this case no ill results ensued from the neglect of measuring the angle delta, but this was due only to fortunate circumstances, and the fact remains that it is important to take both the vertical and the horizontal deviation into consideration.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

THE MYOPIA PROBLEM

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The status of myopia in ophthalmic practice is still uncertain and debatable. No unanimity of opinion exists as to its nature and treatment. In this article I have attempted to review and coordinate the opinions of various writers on the subject and to include some ideas on the problem as seen from my own experience.

A review of the literature seems to show certain rather definite periods of contrasting trends of thought on the subject of myopia. There seems to have occurred a definite evolution from the older mechanistic theory of myopia (elongated globe) toward the more recent biologic variant point of view. It appears difficult for ophthalmologists to discard some of the mechanistic theories. The theories and teachings of the earlier writers taint the therapeutics of myopia even at the present time. Mackenzie [1] (1833), Donders [2] (1864), Landolt [3] (1886), and Beer,[4] who was the first professor of ophthalmology at the University of Vienna, all felt that the prescribing of concave lenses for myopia was wrong, (cited by Jackson [5] and Randall [6]). Perhaps the persistence of this ancient thought accounts for the practice of prescribing partially correcting concave lenses or even of not prescribing lenses at all. The earlier writers were most pessimistic concerning myopia and like Cohn [7] (1867) viewed myopia as a curse of civilization. Cohn

From the Section of Ophthalmology, the Mayo Clinic.

Compiled from a series of lectures given in the Instructional Courses of the American Academy of Ophthalmology and Otolaryngology at Chicago, 1934, Washington, D. C., 1938, and also at Cleveland, Rochester, N. Y., Minneapolis and Topeka, Kan.

1. Mackenzie, cited by Jackson.[5]

2. Donders, F. C.: On the Anomalies of Accommodation and Refraction of the Eye, London, New Sydenham Society, 1864, p. 343.

3. Landolt, cited by Jackson [5]; cited by Randall.[6]

4. Beer, cited by Jackson.[5]

5. Jackson, E.: The Control of Myopia, Am. J. Ophth. **14**:719-725 (Aug.) 1931.

6. Randall, B. A.: Can Hypermetropia Be Healthfully Outgrown? Tr. Am. Ophth. Soc. **26**:657-668, 1890.

7. Cohn, H., cited by Randall [6]; cited by Randall.[9]

wrote extensively and reported the results of his studies of students' eyes. He probably was the first to advocate special help in school and hygienic measures to offset the effects of excessive close work. Harman [8] and others more recently have carried out, with much success, special classes for persons with myopia of the progressive degenerative type. Randall [9] in two papers presented an excellent résumé of the opinions of the earlier writers on myopia. Donders said that the myopic eye was a diseased eye but later changed his opinion and said that myopia was an evolutionary adjustment to environment and that if it were in his power to eradicate all myopia he would not do it. Stilling [10] and others viewed even high degrees of myopia as evidence of national evolution into special fitness for the highest civilization. In contrast to this opinion, hyperopia was thought to be a condition of underdevelopment of lower forms of mankind. Randall thought that hyperopia could not be healthfully outgrown and that to progress from hyperopia to myopia was pathologic. It was also thought that there was no evidence to show that myopia was inherited. Jackson, in a series of scholarly papers, has been outstanding in contributing to the modernization of the conception of myopia and its control. He agreed with Randall that hyperopia cannot be healthfully outgrown and with Harlan [11] that "excessive convergence without accommodation is the most important factor in progressive myopia." Foerster [12] (1886) noticed that many who persistently from youth wore overcorrecting lenses failed to show increase of myopia. He reported 51 cases in which myopia was treated in this manner. Thus it was that at this time the idea of fully correcting myopia with lenses was conceived. This, indeed, was an important mile post. In 1902 Duane [13] in a classic paper stated that the right glasses (full correction) would do more to prevent the progress of myopia than any other procedure. He also advocated exercise and proper posture, such as frequently looking away from the printed page, and also periodic

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8. (*a*) Harman, N. B.: An Analysis of Three Hundred Cases of High Myopia in Children, with a Scheme for the Grading of Fundus Changes in Myopia, Tr. Ophth. Soc. U. Kingdom **33**:202-220, 1913. (*b*) Harman, N. B.; Sorsby, A., and Henderson, T.: Discussion on the Problem of Myopia, ibid. (pt. 2, 1937) **57**: 366-412, 1938.

9. Randall, B. A.: The Refraction of the Human Eye: A Critical Study of the Statistics Obtained by Examinations of the Refraction, Especially Among School Children, Am. J. M. Sc. **90**:123-152, 1885; footnote 6.

10. Stilling, cited by Randall.[9]

11. Harlan, G. C.: Constant Correction of High Myopia, Tr. Am. Ophth. Soc. **28**:373-387, 1892.

12. Foerster: On the Influence of Concave Glasses and Convergence of the Ocular Axes in the Increase of Myopia, Arch. Ophth. **15**:399-435, 1886.

13. Duane, A.: Some Considerations on the Hygienic and Prophylactic Treatment of Myopia, New York State J. Med. **75**:983-986 (June 7) 1902.

examinations in order to bring the glasses up to full correction. He also advocated moderate restriction of near work and the encouragement of out-of-door exercise. Hallett,[14] Pollock,[15] Dobrowolsky,[15] Schiess,[15] Derby,[15] Koster [15] and others proposed the prolonged use of atropine and the cessation of near work. This treatment was supposed to hold the development of the eyes in check and reflexly prevent overconvergence. Lancaster [16] suggested atropinization or occluding of one eye as suggested by Luedde.[17] Morse [18] stressed the elimination of focal infection and endocrine dysfunction, together with periodic examination of the eyes and control of school and home work. He also suggested more didactic work. Improper posture and bending the head forward were held to be mechanical factors in the production of myopia by Lipschutz [19] and Levinsohn,[20] who said that they produced a venous congestion by compressing the posterior ciliary vessels and that they caused ocular malnutrition and orbital congestion. The changes seen about the nerve head in myopia were emphasized by Duane and Jackson as the essential pathologic lesions of myopia. Duane called these changes "sclerochoroiditis posterior." It is to be noted that these same changes are to be seen in the fundi of hyperopic eyes, which Jackson interpreted as eyes in which the progress to myopia had been arrested.

Sourasky [21] in 1928 introduced a somewhat different interpretation of myopia. He expressed the opinion that hyperopia was the normal protection of children's eyes against myopia. He regarded myopia as an abnormal tendency to reduce unduly this "hyperopic reserve" of childhood. This is a modern restatement of Randall's views that hyperopia cannot be healthfully outgrown. Sourasky studied the growth and development of myopia and hyperopia in 621 children of grade school age. Hyperopia increased in 6 per cent, showed no change in 64 per

14. Hallett, De W.: The Prevention of Myopia, Am. J. Ophth. **14**:143-146 (Feb.) 1931.

15. Cited by Hallett.[14]

16. Lancaster, W. B., in discussion on Jackson: The Control of Myopia, J. A. M. A. **105**:1412-1416 (Nov. 2) 1935.

17. Luedde, W. H.: Monocular Cycloplegia for the Control of Myopia, Am. J. Ophth. **15**:603-610 (July) 1932.

18. Morse, S.: Myopia as a Disease Which May Be Preventable, Am. Med. **34**:115-121 (Feb.) 1928.

19. Lipschutz, H.: Myopia and Nearwork, Brit. J. Ophth. **19**:611-612 (Nov.) 1935.

20. Levinsohn, G.: Zur Kritik der Myopiegenese, Med. Klin. **24**:1707-1708 (Nov. 2) 1928.

21. Sourasky, A.: Race, Sex and Environment in the Development of Myopia (Preliminary Communication), Brit. J. Ophth. **12**:197-212 (April) 1928; The Growth of the Eye and the Development of Myopia: A Study in the Changes of Refraction During the School Period, ibid. **12**:625-644 (Dec.) 1928.

cent and decreased in 34 per cent. The degrees of myopia did not change in 34 per cent; there was an increase of less than 1 diopter in 17 per cent, an increase of 1 to 2 diopters in 23 per cent and an increase of 2 to 4 diopters in 16 per cent of the remaining children. Thus, in one large group the myopia was stationary and in a slightly larger group there was only moderate progress of the myopia. In about 6.5 per cent of the children the condition was severe. Sourasky also reported a study of the role of race, sex and environment in the development of myopia, a comparative study of the eyes of boys and girls in Jewish and gentile night schools in London. In these schools the pupils, especially the Jewish boys, were doing excessive near work in an unhygienic environment. He showed that from the time the pupils first entered school the incidence of myopia was much greater among the Jewish group than it was among the gentile pupils. There was little increase in either group during the school age. There was little change in the degree of myopia during the early years in school, although the Jewish children did much finer work. In both groups myopia was twice as frequent among boys as it was among girls, although the Jewish boys did much more exacting work. The author concluded that the higher incidence of myopia among Jewish boys was due to a racial factor and a sex determined factor rather than to excessive close work. He also pointed out the high incidence of low hyperopia among Jewish children, or a lack of hyperopic reserve.

Parsons[22] in 1933 still further modernized the conception of myopia. He said that too much stress is likely to be placed on the length of the eye, and he pointed out that the length of the emmetropic eye may vary as much as 2 mm. and the corneal curvature from 6 to 8.5 mm. without affecting the refractive error. He regarded myopia statistically from the point of view of frequency of distribution. Although emmetropia is the optical ideal normal, the ideal statistical normal varies on either side from emmetropia. These variations toward low hyperopia and low myopia are probably normal. The biologic normal for man has shifted from hyperopia toward emmetropia and low myopia. Many examples of permanently low hyperopia and myopia are seen. These are no more pathologic than short men or tall men. Stationary developmental myopia of 1 to 2 diopters is normal on the other side of the frequency curve of refractive errors. Parsons said that too much emphasis has been placed on visual acuity when visual capacity is the factor which counts most. Visual capacity is made up of visual acuity, field of vision, rapidity of accommodation, adaptation and other factors. Even when visual capacity has decreased one may continue to be efficient

22. Parsons, J.: Developmental Myopia and the Treatment of Myopes, Lancet 2:795-797 (Oct. 7) 1933.

owing to acquired skill. Parsons expressed the opinion that there are many occupations in which a low degree and even 4 and 5 diopters of stationary myopia are an advantage. Persons who have such a degree of myopia do not appear to suffer in any way as a result of near work. He said that there is too much pessimism regarding the effects of near work, and he doubted that it had any appreciable effect in those cases in which the condition progressed. The same author saw real use for special classes for patients who had really progressive myopia but warned against extremes and overenthusiasm. After all, persons who have myopia must work to earn a livelihood and cannot all go to the Riviera to live. He stressed the difference between the developmental and the progressive types of myopia.

Sorsby [23] attacked the elongated globe or axial theory of myopia and supported the binomial variation theory of Tron [24] and Steiger.[25] Sorsby pointed out that the length of the globe was significant only in high hyperopia and in high myopia of more than 6 diopters. He pointed out that axial length may be the same in a great variation of refractive errors, that is, from +6 diopters to —9 diopters. He said that low and medium errors of refraction should be regarded as biologic variants and not as pathologic entities. According to Sorsby, the emmetropic eye should no longer be regarded as the ideal and departures from it as errors. The biologic mathematical approach, he thought, broke the idea of axial length as the exclusive cause of myopia and the fallacy that every eye on the minus side of emmetropia was pathologic. He expressed the opinion that myopia is due to heredity, a biologic variable characteristic, and to the use of the eyes (environment). Sorsby said that a change in the conception of myopia had occurred in recent years and that the approach to the problem has changed from the mechanistic to the biologic. This has evolved a method of study rather than a solution of the problem. Sorsby recognized two types of myopia: (1) a physiologic variant and (2) a pathologic process. This new point of view, he thought, denied all of the cherished dogmas of the past century and theoretically established what most ophthalmologists had learned clinically, namely, that most myopia is not pathologic. Myopia had emerged as a problem in biology. Sorsby contended that Helmholz' dictum that the eye is a most imperfect instrument is after all but the admission that Helmholz' contemporaries were unable to understand anything that was not mechanical.

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23. Sorsby, A.: The Control of School Myopia, Brit. M. J. **2**:730-733 (Oct. 21) 1933; The Pre-Myopic State: Its Bearing on the Incidence of Myopia, Tr. Ophth. Soc. U. Kingdom **54**:459-465, 1934.

24. Tron, E., cited by Sorsby.[23]

25. Steiger, A., cited by Sorsby.[23]

Henderson [26] regarded axial myopia as the result of constitutional factors such as inadequate scleral tissues causing the posterior pole of the globe to give way under normal intraocular pressure ("scleral rickets"). He expressed the opinion that hyperopic eyes are undersized and underdeveloped, whereas myopic eyes are oversized and overdeveloped. He said that ill health and lowered vitality contribute to weakness of the supporting framework of the eye during growth just as they contribute to the production of flatfoot. He stated that correction of the refractive error is only a treatment of the effects of myopia. He denied the idea that there was a physiologic myopia any more than there was a physiologic form of flatfoot. He saw no reason for a person wearing glasses for low degrees of myopia, that is, 2 to 3 diopters, even if the affected person was a child. Thus the argument as to the nature of myopia continues throughout the literature.

Haseltine [27] thought that glaucoma and myopia might have the same origin but might differ in their manifestations because of difference in elasticity of the tissues of the eye, that is, the more elastic and distensible eyes might stretch and produce myopia. Wiener [28] cited von Arlt's theory that increased tension by use of the accommodation might be a factor in producing myopia, but he also said that Hess and Heine had found that this was not true.

Haseltine administered powdered extract of the adrenal gland and thought that it improved the physical state and scleral tone. Bothman [29] and Costello [30] both noted that myopia was often seen among children who had hypothyroidism. These authors thought that there might be some relation between decreased thyroid secretion and myopia. Both reported that the oral administration of thyroid extract favorably influenced the progress of myopia in cases of hypothyroidism. Walker [31] and Law [32] suggested calcium deficiency as a factor in myopia and

26. Henderson, T.: The Constitutional Factor in Myopia, Tr. Ophth. Soc. U. Kingdom **54**:451-459, 1934.

27. Haseltine, S. L.: Suprarenal Gland in the Treatment of Glaucoma, Progressive Myopia, and Some Allergic Conditions, J. M. Soc. New Jersey **34**:729-731 (Dec.) 1937.

28. Wiener, M.: (*a*) Use of Epinephrine in Progressive Myopia, Tr. Sect. Ophth., A. M. A., 1930, pp. 256-277; (*b*) Myopia: Cause, Progress and Treatment, South. M. J. **24**:529-534 (June) 1931.

29. Bothman, L.: The Relation of the Basal Metabolic Rate to Progressive Axial Myopia, Am. J. Ophth. **14**:918-924 (Sept.) 1931.

30. Costello, J. P.: Obesity and Ocular Symptoms in Mentally Alert Children Due to Hypothyroidism, Endocrinology **20**:105-106 (Jan.) 1936.

31. Walker, J. P. S.: Progressive Myopia, a Suggestion Explaining Its Causation, and for Its Treatment, Brit. J. Ophth. **16**:485-488 (Aug.) 1932.

32. Law, F. W.: Calcium and Parathyroid Therapy in Progressive Myopia, Tr. Ophth. Soc. U. Kingdom **54**:281-290, 1934.

administered calcium and parathyroid extract by mouth. They stated that no benefit would be obtained by using calcium alone. Knapp [33] discussed vitamin D and calcium deficiency as factors in producing myopia. He claimed that the administration of calcium and vitamin D produced improvement in some cases. Laval [34] disagreed with those who thought that treatment with vitamin D and calcium helped patients who had myopia.

Wiener advocated the instillation of a solution of epinephrine in the conjunctival sac to check the progress of myopia. He called attention to the theory of Ince, that myopia was due to tissue asthenia, and the theory of Levinsohn, that this asthenia was due to decreased secretion by the adrenal glands, possibly because of improper posture and lack of exercise. Wiener also cited Pontius, who said that epinephrine was beneficial in cases of corneal ectasia and that the cornea and sclera are the same tissues embryologically. In 159 cases in which myopia was treated with exercise, correction of posture and the administration of epinephrine, Wiener noted that the condition was checked in 79 cases and that in 20 cases it had progressed less than 0.25 diopter in any one year. Wiener concluded that heritage or a congenital predisposition is the chief factor in the production of myopia just as it is in stature. The use of epinephrine was also advocated by Hird,[35] Dvorak [36] and Malone.[37] Hird said that for the past four years he had prescribed a solution of epinephrine hydrochloride (1:1,000), which was instilled into the eye three times a day. In 80 per cent of the cases in which follow-up data were available this treatment checked the progress of myopia.

Marlow [38] expressed the opinion that muscle imbalance is probably the most important factor in the progress of myopia. He said that an occlusion test should be carried out as early as possible. He recommended the prescribing of prisms for latent exophoria and hyperphoria to aid in decreasing or checking myopia. Snell [39] found that the dynamic powers of convergence are maintained in cases of myopia. In 90 per cent of 100 consecutive cases the average amplitude of convergence was found to be greater than normal. Dvorak advocated the constant use of full cycloplegic correction, the use of prisms for hyperphoria and

33. Knapp, A.: Personal communication to the author.

34. Laval, J.: Vitamin D and Myopia, Arch. Ophth. **19**:47-53 (Jan.) 1938.

35. Hird, R. B., in discussion on Harman, Sorsby and Henderson.[8b]

36. Dvorak, J. E.: The Present Status of Management of Myopia, J. Iowa M. Soc. **26**:25-31 (Jan.) 1936.

37. Malone, J. F.: Unpublished data.

38. Marlow, F. W.: Muscle Imbalance in Myopia, Arch. Ophth. **13**:584-597 (April) 1935.

39. Snell, A. C.: A Statistical Study of Functional Muscle Tests in Axial Myopia, Tr. Sect. Ophth., A. M. A., 1936, pp. 49-64.

exophoria and the use of a solution of epinephrine. He also advised physical exercises, hygienic measures, proper reading habits and myope classes for patients who had obstinate and degenerative myopia. Jackson [40] expressed the opinion that the progress of myopia in early life can be checked by the use of fully correcting lenses, proper posture and the correction of habits of excessive convergence.

Ball [41] reported that the Abbé Desmonceaux (1734) was the first to suggest and carry out the removal of the crystalline lens for the relief of myopia. O'Connor [42] reported 6 cases of myopia in which removal of the lens proved successful. The patients were carefully selected, and capsulotomy and repeated needlings were done. Elschnig [43] reported 110 cases of myopia in which the lens was removed. Loss of the vitreous occurred in only 5 per cent of these cases. He said that operation properly done did not increase the liability to detachment of the retina. He stated that the myopia should be 18 diopters or more, otherwise there would be a postoperative hyperopia of 5 diopters and not much would have been gained.

Parsons classified myopia on an etiologic basis as follows:

1. Abnormally increased length of the globe, axial myopia
2. Abnormal curvature of the refracting surfaces, curvature myopia
   (*a*) Too strong curvature of the cornea
   (*b*) Too strong curvature of one or both surfaces of the lens
3. Abnormal refractive indexes of the media, index myopia
   (*a*) Too high index of cornea or aqueous
   (*b*) Too high total index of lens, owing to too high index of nucleus or too low index of cortex
4. Displacement forward of lens
5. Combinations of these factors

Such a classification is intelligible, inclusive and workable, especially for statistical and filing purposes. I should like to suggest the following clinical grouping:

1. Developmental myopia
   (*a*) Curvature myopia
   (*b*) Axial myopia
2. Malignant (degenerative or deteriorative myopia)
   (*a*) Moderate degree
   (*b*) Severe degree

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40. Jackson, E.: The Control of Myopia, J. A. M. A. **105**:1412-1416 (Nov. 2) 1935.

41. Ball, J. M.: The Abbé Desmonceaux and Removal of the Lens for Myopia, Am. J. Ophth. **11**:223-224 (March) 1928.

42. O'Connor, R.: Lens Removal for High Myopia: Results in Ten Eyes, Am. J. Ophth. **16**:516-518 (June) 1933.

43. Elschnig, A.: Lens Extraction, Am. J. Ophth. **17**:1118-1121 (Dec.) 1934.

Curvature myopia is the type seen in 75 per cent of cases. It is innocent and is arrested early. Axial myopia occurs in 20 per cent of cases. It also is innocent and is arrested early. This type is similar to a mild form of malignant myopia; that is, it is associated with some changes in the vitreous and a mild degree of stretching of the retina and choroid. Malignant, or degenerative, myopia is distinctly pathologic. A moderate degree of malignant myopia occurs in 4.5 per cent of cases and a severe degree in 0.5 per cent of cases of myopia.

Myopia constitutes a small minority of the refractive errors seen in private practice. In discussing the incidence of refractive errors among newborn infants, Wiener [28b] said: "A summary of 2,400 eyes reveals 9 myopes, 21 emmetropes and 2,370 hyperopes." Hess,[44] who examined the eyes of a group of children in country schools, found that 15.1 per cent were myopic, 33.2 per cent were emmetropic and 51.7 per cent were hyperopic. Among primitive people, such as American Indians and the Negro race, the incidence of myopia is low, that is, about 2 or 3 per cent. Rasmussen [45] said that more than 50 per cent of the Chinese children in mission schools had myopia. He expressed the opinion that the use of fully correcting lenses tended to reduce the degree of myopia. Mills [46] said that of his patients who were artists 48 per cent were myopic. He expressed the opinion that myopia would have considerable effect on the principles and interpretation of art. Banerjee,[47] of India, said that 20 per cent of his patients were myopic. It is well known that in cases of myopia there is a definite and marked increase in the degree of myopia while the affected persons are in high school and college and are using their eyes intensively for close work. It is also admitted that in many cases myopia ceases to progress during the time the affected persons are in high school and college. Although a great amount of research has been done on myopia and the management of this condition has been greatly improved, the incidence of the condition does not appear to be increasing or decreasing.

In the great majority of cases of myopia the condition seems to be a developmental defect, a deviation from the statistical normal in frequency distribution of refractive errors, such as is seen in stature. Von Arlt [48]

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44. Hess, cited by Wiener.[28b]

45. Rasmussen, O. D.: Incidence of Myopia in China: Data and Theses from Periodical Investigations Covering Thirty Years Residence, and Association with Refracting and Hospital Centres, in a Score of the Larger Cities, Brit. J. Ophth. **20**:350-360 (June) 1936.

46. Mills, L.: Peripheral Vision in Art, Arch. Ophth. **16**:208-219 (Aug.) 1936.

47. Banerjee, J.: State of Vision of Indian Students in Calcutta, Calcutta M. J. **33**:53-63 (Feb.); 285-295 (June) 1938.

48. Von Arlt, cited by Wiener.[28b]

and Steiger [49] said that "the natural development of the eye must lead to the refractive condition to which it was potentially directed at conception." As Parsons suggested, the biologic normal in man has probably shifted from hyperopia toward emmetropia and myopia. A permanent low degree of myopia is no more pathologic than is hyperopia of the same degree. As in all other fields of medical diagnosis, the limits of normal widen and the limits of the pathologic condition narrow with increased understanding of the condition. It is probable that the only cases of myopia in which it is necessary to adopt stern measures of treatment are the relatively few cases in which the condition gets beyond control.

As indicated by a review of the literature, the cause of myopia is still in doubt. The excellent research work of Sourasky seems to throw some light on this aspect of the subject. Myopia may be regarded as an abnormality which tends to reduce the hyperopic reserve of childhood to the myopic side. The normal tendency is for hyperopia to remain stationary throughout school life. Any noticeable reduction of hyperopia in this period is abnormal. Such reduction is the root of myopia, and, if the reduction is marked, myopia must supervene unless there is a high hyperopic reserve. The reduction may be marked or feeble; it affects both hyperopes and myopes alike and is noticeable only in the latter because of reduced visual acuity. The process may become arrested at any stage (stationary) or may continue unchecked (malignant). The process is not myopia, per se. The myopic eye becomes elongated only because it is subjected to this affliction. Myopia is no more the cause of elongation of the globe than a glaucomatous cup is the cause of increased tension. Myopia is the result and not the cause of such an abnormal tendency.

In trying to arrive at the most plausible cause of myopia, heritage seems to be the one outstanding factor. Myopia is undoubtedly an inherited characteristic, which, in all probability, follows Mendel's law. The factor of sex determination seems also to be of importance, particularly among some races, such as the Jewish race. That an endocrine disturbance may well play a part is strongly suggested by the onset and progression of myopia in childhood and adolescence, but this is unproved. To one who has seen many cases of myopia and obtained follow-up data for a period of years, it is difficult to discount entirely the effects of excessive near work under poor hygienic conditions. In many cases the myopia has progressed under these circumstances. Excessive near work cannot be blamed too strongly, however, for myopia often becomes arrested at the age when most near work is being done (while the affected persons are in high school and college). Other factors, such as faulty

49. Steiger, cited by Wiener.[28b]

posture, poor lighting and scleral ectasia, are of debatable significance but still must be given serious consideration. Age is, of course, a notable factor, as true myopia occurs and develops during childhood and adolescence.

In the management of myopia ophthalmologists are chiefly concerned with childhood and adolescence. The curvature and axial forms of developmental myopia are considered much alike, except that somewhat more concern is felt over the latter (axial) type because of its miniature likeness to the malignant forms of the disorder. Tendency to opacities in the vitreous, small posterior staphyloma and thinning of the posterior portion of the uvea make one think that one may be dealing with mild deteriorative myopia. Of all measures that have been advocated, I feel that fully correcting lenses, constantly worn, are the most important. It is my feeling that when I have been able to treat myopia from its incipiency with fully correcting lenses, constantly worn, a normally functioning pair of eyes has been obtained. In cases of myopia in which the patients are attending school, it has been the routine at the Mayo Clinic to examine the patients during the Christmas, Easter and summer vacations. In this way it has been possible to keep the correcting lenses apace with any changes that occurred in the eyes. Too much stress, I feel, cannot be placed on the importance of regular and frequent examination. The work of the public health service and visiting nurse in detecting beginning myopia and referring the affected persons for treatment is to be highly commended. It would seem that all young persons who are attending grade school or high school should have their vision tested regularly in order to determine whether they need assistance. Although the relation of near work to the production of myopia is questionable, it would appear sensible to eliminate useless and unnecessary close application of the eyes. All ocular work not necessary to the education of the child might well be dispensed with until the status of the myopia has been established. The preponderance of sound clinical evidence in the literature forces one to consider seriously the effects of improper posture, poor reading habits and factors contributing to excessive convergence. General hygienic and dietary measures would seem feasible. Out-of-door exercise could well be substituted for unnecessary indoor pastimes demanding the use of the eyes in near vision.

In the rare cases in which the myopia is malignant or distinctly pathologic, the problem becomes an individual one. In these cases the myopia must be managed according to the medical, sociologic and economic background. After all, in most of these cases the patients must work in order to live. Common sense must be used in the advice given them. It is a serious matter to advise one to change his environment and occupation. The ophthalmologist should be sure that it is necessary. Perhaps as much could be accomplised by carefully planned regulation of near work

and general living conditions at home. Just recently I saw a patient who had typical high myopia which had remained stationary for thirty-six years. The myopia had all the marks of the malignant type, including posterior staphyloma, a thinned posterior uvea and a high refractive error. The distance correction which had been prescribed originally was still correct, and he came in merely to obtain presbyopic correction. One who originally sees such a patient at the age of 14 years could easily have become unduly concerned as to the progress of the myopia, but in this case the eyes are still functioning normally. Undue pessimism, even regarding the malignant type of myopia, is not indicated. Sensible regulation would seem much more useful. Early, full correction by lenses is essential in my opinion.

Various theoretic therapeutic measures may be tried. Prolonged atropinization and rest have been advocated. It was thought that suspension of the accommodation reflexly prevents overconvergence. This enforced rest of the eyes might be used during vacation periods in cases of apparently progressive myopia. However, it also has been argued that a normally functioning accommodative mechanism is essential in the prevention of the development of myopia. Such is the theory behind the use of fully corrective lenses. Atropinization or the occlusion of one eye might be tried for its effect on lessening of convergence. In cases of progressive myopia in which the basal metabolic rate is low, Bothman advocated the use of thyroid extract. The oral administration of calcium and parathyroid extract was used by Walker. In giving calcium by mouth the drug must first be dissolved in a warm solution; otherwise it is not absorbed and remains inactive. Walker said that no action was obtained by the use of calcium alone. Wiener said that the instillation of epinephrine into the conjunctival sac arrested the progress of the myopia in 80 per cent of 99 cases, and this treatment may well be worth trying. For a number of years I have been using instillation of a solution of epinephrine hydrochloride (1:1,000) for a series of children with borderline progressive axial myopia. For some, it appeared to affect favorably the progress of the myopia, but this is difficult to prove. Jackson, however, obtained equally satisfactory results (arrest of the myopia in 79 per cent of cases) by the use of fully correcting lenses and correction of factors leading to excessive convergence. The use of "base in" prisms incorporated in the correcting lenses has been advocated, and this might be tried in an atttempt to lessen the convergence.

The question of whether to undercorrect, to correct fully or to overcorrect the myopia by lenses is most interesting. The question is the same whether cycloplegia is used or not, for the use of cycloplegic drugs does not necessarily prevent acceptance of overcorrecting lenses. When concave lenses are placed in front of the eyes and the point of correction is being approximated, one of several images may be selected by the

patient as the best. This may vary from slight undercorrection to definite overcorrection and is particularly true when astigmatism is present and the interval of Sturm is involved. By studying the images of Sturm's interval one can see that considerable allowance can be had in selection of the best image near the point of correction. To say just which correcting lens or image is the exact correction involves much speculation. I know that I definitely used overcorrection in many cases of myopia in former years. The patients as a group were comfortable and enjoyed good vision, and normally functioning eyes developed. In recent years I have tried to hold correction down to exact full or slight undercorrection. In cases in which this practice was followed the patients have not been as comfortable and have complained of slightly blurred distant vision, and I believe that the myopia has shown more tendency to progress. Personally, I prefer ample full correction. This allows the ciliary mechanism to assume a more active tone, which resembles that of the hyperopic eye. The patients enjoy the improved distant vision. It is possible from these observations that the myopia has shown less tendency to progress. This, however, is not proved. I believe that the same correction should be worn for both distant and near vision in eyes not presbyopic. In order to keep the ciliary mechanism functioning properly and the extraocular muscle balance in correct ratio, it would seem that the full correction is equally essential in near vision. There are, of course, cases of extremely high myopia in which full correction is not feasible or comfortable, the same as in high degrees of hyperopia. This applies also to very high degrees of astigmatism. The correction in these cases is a matter of trial and judgment. A reduced correction, equivalent formulas being used, is often useful, as I have pointed out in a previous paper.[50]

CONCLUSIONS

1. The mechanical theory (elongated globe) of myopia is untenable. Current opinion has shifted to a biologic point of view regarding the frequency distribution of myopia in refractive states.

2. In the majority of instances myopia is not abnormal but rather a variant of the type of refraction of the eye.

3. Myopia is a biologic tendency to reduce the hyperopic reserve usually seen in childhood.

4. This tendency is seen in association with hyperopia and myopia but is troublesome only in cases of myopia.

5. This process may become arrested at any stage or continue unchecked.

6. Myopia is the result, not the cause, of this process.

50. Prangen, A. de H.: Some Problems and Procedures in Refraction, Arch. Ophth. **18**:432-447 (Sept.) 1937.

7. An adequate hyperopic reserve in young persons seems essential as a protection against the development of myopia.

8. Children who have low hyperopia should be watched. They may not be as normal as they commonly are considered to be.

9. A hereditary tendency, together with age, race, and sex, is the chief etiologic factor. The importance of all other factors is debatable, but those contributing to excessive convergence must be considered seriously.

10. Constant wearing of fully correcting lenses is of paramount importance.

11. Periodic examinations to keep the correcting lenses apace with any change in the myopia are a necessity.

12. General hygienic and dietetic measures are indicated.

13. Restriction of unnecessary near work by children seems sensible.

14. Instillation of epinephrine is of doubtful value but is worth trying. This may also be true of calcium, parathyroid extract, vitamin D and thyroid extract.

15. Progressive myopia of the malignant type is rare. It is an independent biologic group. Its management and treatment are individual problems.

News and Notes

GENERAL NEWS

The Herman Knapp Memorial Eye Hospital.—The Herman Knapp Memorial Eye Hospital at 500 West Fifty-Seventh Street, New York, will be taken over by the Columbia-Presbyterian Medical Center and Columbia University on Jan. 1, 1940. The Hospital will close on that date, and the hospital activities and the care of the outpatient department will be continued by the Institute of Ophthalmology of the Presbyterian Hospital and the Vanderbilt Clinic. The assets and funds of the Knapp Memorial Eye Hospital, including the present building, will go to Columbia University to found the Knapp Memorial Foundation in Ophthalmology. The income is to be used for study, postgraduate teaching and fellowships. The foundation will be directed by a committee consisting of Dr. Arnold Knapp, Dr. Phillips Thygeson, executive officer of the department of ophthalmology of Columbia University, and Dr. Willard C. Rappleye, dean of the Columbia University College of Physicians and Surgeons.

The Herman Knapp Memorial Eye Hospital was founded by the late Dr. Herman Knapp in 1869 and was then known as the New York Ophthalmic and Aural Institute, at 46 East Twelfth Street. During its first year the institute cared for 1,828 patients in the dispensary and 143 resident patients. In addition, instruction was given to 46 students (ARCH. OPHTH. **14**: 909 [Dec.] 1935).

The institute remained at 46 East Twelfth Street for forty-three years. An addition was built in 1879 to make room for a laboratory, which also served as a lecture room. In 1894 the adjoining house, at 44 East Twelfth Street, was acquired and remodeled as an outpatient department, with optician and drug room facilities and a clinical operating room.

In 1909 Dr. Herman Knapp was succeeded as executive surgeon by his son, Dr. Arnold Knapp. In 1913 the institute was moved to Fifty-Seventh Street and Tenth Avenue; its name was changed to the Herman Knapp Memorial Eye Hospital, and the aural department was abandoned. In addition to its regular medical work and postgraduate teaching, most of the instruction in ocular diseases was given there to the students of the Columbia University College of Physicians and Surgeons, until the college moved to its new site in 1928.

Though the work of the hospital in its Fifty-Seventh Street location was most satisfactory, the trustees of the hospital have realized for some time that the future of the smaller specialty hospitals in New York is a hazardous one, and after careful consideration they came to the conclusion that close association with a large general hospital and a university medical school was essential.

This institution is completing its seventy years—forty-three years at 46 East Twelfth Street and twenty-seven years at 500 West Fifty-Seventh Street. In this period 800,000 patients have been treated, 730,000 in the outpatient department without charge and 70,000 inpatients, of whom 76 per cent were treated free. While the cessation

of this hospital in its present location was decided on only after the most careful deliberation, the trustees feel that in the coming merger with the Columbia-Presbyterian Medical Center a forward step may be heralded and the original ideas of the founder, Herman Knapp, carried out in adequate and permanent form.

Clinical Course in Ophthalmology and Otolaryngology.—The ninth annual midwinter clinical course in ophthalmology and otolaryngology given by the Research Study Club will be held in Los Angeles between Jan. 15 and 26, 1940. The principal teachers in the ophthalmic lectures will be Dr. Albert D. Ruedemann, of Cleveland; Dr. Algernon B. Reese, of New York and Dr. Meyer Wiener, of St. Louis. Registration may be made by sending a check for $25 to Dr. Pierre Violé, treasurer, 1930 Wilshire Boulevard, Los Angeles.

Ophthalmic Award.—A gold medal is awarded annually by the University of Buffalo for work on an ophthalmologic subject. Further information regarding this award may be procured from Dr. H. W. Cowper, 543 Franklin Street, Buffalo.

Presentation of Portrait of Dr. Edward Jackson.—At a formal ceremony held at the Wills Hospital in Philadelphia on October 19 a portrait of Dr. Edward Jackson was presented to the hospital by Dr. William Zentmayer, of Philadelphia, in behalf of the Dr. Edward Jackson Portrait Fund Committee. Dr. Milton Griscom, chief of staff of the Wills Hospital, was in charge of the program and arrangements. Mr. Ernest T. Trigg, of Philadelphia and Washington, received the portrait for the hospital on behalf of the Wills Hospital Committee of the Board of City Trusts of the City of Philadelphia.

In presenting the portrait, Dr. Zentmayer said:

> It is fitting that a portrait of Dr. Jackson, the gift of his loyal friends throughout the country, should be brought to Philadelphia, as it was in this city that Dr. Jackson laid the foundation of his splendid career that has made him eminent among ophthalmologists of the world.
>
> It is appropriate that this portrait be presented to the Board of Directors of City Trusts, to be hung on the walls of the Wills Hospital, as Dr. Jackson served the institution faithfully for a number of years and did much to add to its fame. His interest in the hospital has never flagged, and neither distance nor years have deterred him from making almost yearly visits to it.
>
> His many valuable contributions to all phases of ophthalmology will preserve his name in the archives of medicine, but it is his unselfish devotion to the promotion of higher standards in ophthalmology, his enduring interest in the younger men and his wise counsel and kindliness that endear him to his contemporaries and friends.
>
> In behalf of the committee, I now present this portrait of Dr. Jackson—physician, humanitarian, leader, teacher, author and editor.

With a few gracious words, Mr. Trigg accepted the portrait on behalf of Wills Hospital Committee of the Board of Directors of the City Trusts and emphasized what Dr. Jackson's connection with the Wills Hospital meant to the institution.

Dr. Jackson made the following reply:

It is very pleasant to come back to the Wills Hospital, especially to see its new and well planned home and chiefly because of the memories it holds. Of the first four surgeons at the hospital, I remember the face and figure of Squier Littell. Isaac Hays was still living but never visited the hospital. He was best known to me by the *American Journal of Medical Sciences,* which he edited for fifty years, and by his copies of the early volumes of the *Transactions of the American Ophthalmological Society,* of which society he was an honorary member from its beginning.

When I was a medical student, the four surgeons at the Pennsylvania Hospital had all been surgeons at the Wills Hospital. They were D. Hayes Agnew, William Hunt, Richard J. Levis and T. G. Norton. Edward C. Ellett, who last week received the Leslie Dana Medal, awarded for achievement in the prevention of blindness, and Walter R. Parker, for many years professor of ophthalmology in the University of Michigan, were the residents when Samuel D. Risley, Charles A. Oliver, Frank Fisher and myself were elected to the surgical staff. George C. Harlan, my former chief, and William F. Norris, with whom William Zentmayer was working, were still serving on that staff. This list of names will suggest some of the memories associated with the Wills Hospital. The honor of this ceremony will remain among its most pleasant recollections.

Cumulated Index of the Archives of Ophthalmology.—Requests have been received for a ten year index of the ARCHIVES OF OPHTHALMOLOGY. Before serious consideration is given to the production of a cumulated index, it is desirable to know whether the demand for it would be sufficient to warrant its sale at not to exceed $5 a copy; that is, whether one thousand copies could be sold. It will be appreciated if those who are interested in such an index will fill out and send the form which appears below to the Managing Editor at the publication office, 535 North Dearborn Street, Chicago.

I SHOULD BE WILLING TO SUBSCRIBE TO A CUMULATED INDEX OF THE ARCHIVES OF OPHTHALMOLOGY AT $5.00.

NAME .....

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DATE.....

Leslie Dana Gold Medal.—The Leslie Dana Gold Medal, awarded annually for "outstanding achievements in the prevention of blindness and the conservation of vision," was presented to Dr. Edward C. Ellett, of Memphis, Tenn., at a dinner in his honor at St. Louis on Saturday evening, October 14. The presentation was made by Dr. Edward Jackson, of Denver, who was the first recipient of the Leslie Dana Medal in 1925.

Dr. Ellett, who is 69 years old, has practiced ophthalmology in Memphis since 1893. He was professor of ophthalmology at the University of Tennessee from 1906 to 1922. He is a former chairman of the Section on Ophthalmology of the American Medical Association, a former president of the American Academy of Ophthalmology and Otolaryngology and a former president of the American Ophthalmological Society.

The inscription on the 1939 medal refers to Dr. Ellett as an "inspiring teacher, skilled surgeon, understanding and sympathetic clinician and friend."

The conditions of the award of the Leslie Dana Medal set forth that it is to be made for "long meritorious service in the conservation of vision in the prevention and cure of diseases dangerous to eyesight; research and instruction in ophthalmology and allied subjects; social service for the control of eye diseases; and special discoveries in the domain of general science or medicine of exceptional importance in conservation of vision."

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Aqueous Humor

THE THEORIES ON THE FORMATION OF THE AQUEOUS HUMOUR. J. D. ROBERTSON, Brit. J. Ophth. 23: 243 (April) 1939.

Robertson discusses the fundamentals in connection with the problem of the formation of the aqueous humor, such as the nature of crystalloids and colloids and the permeability of membranes. Definitions are given of the terms dialysates, ultrafiltrates, exudates, transudates and secretion.

After analyzing the evidence bearing on a solution of the problem as to the nature of the aqueous humor, the author states that the evidence on which he rests his belief that the aqueous humor is produced by secretion is based more on physiologic facts than on the anatomic structure of the ciliary processes, cytologic evidence of functional activity, electric evidence and the stimulation of secretion by drugs or by nerves, though in the opinion of many workers these last four methods of investigation in themselves produce strong evidence as to the secretory nature of the intraocular fluid.

W. ZENTMAYER.

Biochemistry

THE OXIDATION-REDUCTION PROCESS IN THE OPHTHALMOPATHIES. A. RIZZO, Ann. di ottal. e clin. ocul. 66:785 (Oct.) 1938.

The glutathione content of the blood was determined for 6 normal persons, aged 54 to 70, for 27 persons with senile cataract, and for 7 persons with chronic glaucoma. The average for the normal persons was 21.1 mg. per hundred cubic centimeters of blood. A definite increase in the glutathione content was found for the patients with cataract, while a slighter increase was found for those with glaucoma. This increase in the oxidative power of the blood may represent a compensatory mechanism made necessary by a decrease in the oxidative capacity of the tissues.

A bibliography is included.

S. R. GIFFORD.

Color Sense

ISHIHARA'S TABLES OF THE SOVIET EDITION. I. BARBEL, Vestnik oftal. 10: 684, 1937.

Barbel made a comparative study of the original Japanese and of the first Russian edition of Ishihara's tables on 300 persons (of these 49 had poor color vision). One hundred and ninety-four trichromates gave identical answers, and only 57 made some mistakes in tables 5, 10, 11 and 12. Identical answers were given by 23 patients with poor color vision, while the other 26 patients gave contradictory answers,

chiefly in tables 6 and 10, as in these the hidden number was not read by 10 people in the Russian edition and was missed by only 2 patients in the Japanese edition.

Barbel considers the Soviet edition of the Ishihara tables to be comparable to the Japanese edition. In doubtful cases he recommends the use of two pigment tests.

O. SITCHEVSKA.

Conjunctiva

STAPHYLOCOCCUS CONJUNCTIVITIS. C. S. O'BRIEN and J. H. ALLEN, Am. J. Ophth. **21**: 641 (June) 1938.

O'Brien and Allen discuss staphylococcic conjunctivitis from the standpoint of pathogenicity as opposed to the older view of these organisms being saprophytes. After a study of 2,110 consecutive cases of conjunctivitis, they found the infection due to staphylococci in almost half the determinable cases. Their observations indicate that the disease is communicable, though it does not appear to be easily transmitted. Its clinical manifestations and treatment are discussed.

W. S. REESE.

MEIBOMIAN CONJUNCTIVITIS CAUSED BY ACROTHECIUM HOMINIS OLAH: REPORT OF A CASE. A. FAZAKAS, Klin. Monatsbl. f. Augenh. **101**: 387 (Sept.) 1938.

Acrothecium hominis Olah is described as the cause of conjunctivitis for the first time. The patient had suffered for a long time. The tarsus and the bulbar portion of the lower lids were greatly thickened. The meibomian tubes were enlarged, some of them presenting small tumors, resembling chalazia. Fistulas had formed into the conjunctiva, out of which a thin discharge could be pressed which contained the aforementioned fungus.

Fazakas describes these myceles, their composition and their conidiophores, which are known to the dermatologist. Keratomycosis was produced successfully in rabbits, with this fungus; its production could also be prevented by the simultaneous internal administration of potassium iodide. Similar experiments proved the toxicity of these myceles to the conjunctiva. The fungus was found in the meibomian tubes, but it was found in the conjunctival secretion only during the acute stage of the infection. A recurrence could be expected whenever the fungus was present in the conjunctival discharge during recovery. Proper therapy, which is not described, succeeded in removing the fungus from the meibomian glands, and in alleviating the chronic irritation.

K. L. STOLL.

Comparative Ophthalmology

ACUTE AMAUROTIC EPILEPSY IN MACACUS RHESUS. L. V. BOGAERT and H. J. SCHERER, Arch. Neurol. & Psychiat. **40**: 521 (Sept.) 1938.

This study on acute amaurotic epilepsy is founded on 16 clinical and 7 pathologic observations. Clinical study showed a disease charac-

terized by blindness of central origin, epileptic fits, disturbance of movement and gait and a marked reaction in the spinal fluid. The disease may cause death or end in complete recovery. The evolution is always acute.

Pathologic study showed a primary cerebral disease occurring in animals which were otherwise healthy; no septic embolic process was observed at autopsy. The peripheral visual paths, spinal cord and peripheral nerves were normal. The cause of the disease remains unknown. An infection as well as an intoxication is possible. A somewhat similar amaurotic epilepsy has been reported in man.

R. IRVINE.

Cornea and Sclera

MALIGNANT SCLERITIS. A. CORRADO, Rassegna ital. d'ottal. **5**: 225 (March-April) 1936.

The author reports a case of diffused malignant scleritis in the left eye of a man 71 years of age. The scleral lesion began 3 to 4 mm. from the limbus on the temporal side. It was a firm tumefaction of rose-wine color. Later nodular growths formed, which became ulcerated. The contents seemed to be caseous. The cornea was normal. There was some uveitis with the scleritis. Microscopic examination of the secretion and inoculation of the rabbit failed to show a pathogenic organism.

Roentgen and clinical examination of the chest showed old sclerotic patches due to healed pulmonary tuberculosis.

The ocular lesion healed in one year, resulting in iridodonesis, subluxation of the lens and scleral ectasia.

A. PERZIA.

HERPES OF THE CORNEA AND THE CLAUDE-BERNARD-HORNER SYNDROME. E. FEDERICI, Rassegna ital. d'ottal. **6**: 3 (Jan.-Feb.) 1937.

The author reports a rare case of corneal herpes associated with the Claude-Bernard-Horner syndrome and tries to trace the relation of the herpes virus to the ocular sympathetic nerves.

In corneal herpes there are nerve contacts between the trigeminal nerve and the sympathetic nerves. The author believes that the virus of herpes ascends the nasal branch of the ophthalmic nerve and is transmitted by a fine branch of the sympathetic nerve and thus produces the Claude-Bernard-Horner syndrome. This is only a supposition, as such cases are rare and in many cases of herpes the symptoms are not a part of the syndrome mentioned.

A. PERZIA.

NONFAMILIAL JUVENILE DYSTROPHY OF THE CORNEA: REPORT OF A CASE. E. SAGHER, Klin. Monatsbl. f. Augenh. **101**: 507 (Oct.) 1938.

Contrary to familial dystrophies of the cornea, the nonfamilial type shows somatic changes which point to developmental or degenerative disturbances. These are enumerated. They may involve the skull and the skeleton or manifest themselves as idiocy, tumors of the hypophysis or lipoid degeneration of the brain or of the optic nerve.

Sagher reports the case of a girl aged 13 years, the youngest of four children, who showed a corneal opacity at the age of 2. The causation was not known, but the girl was underdeveloped and underweight. Her skull and skeleton were normal, and the liver and spleen were normal in size and in function. Vision was reduced to 6/18 in the right eye and to 6/36 in the left eye; the intraocular tension was increased, and parenchymatous opacities existed without involvement of the epithelium or of Descemet's membrane. The fundus was normal and did not present any glaucomatous changes. The blindspot was of normal outline, and the field of vision showed a normal periphery. Treatment was of no avail. The corneal disturbance resembled those accompanying gargoylism or dysostosis multiplex. The presence of choreatic twitchings corroborated the opinion that the corneal dystrophy was due to a developmental disturbance of a degenerative nature. K. L. STOLL.

Experimental Pathology

EYE LESIONS IN EXPERIMENTAL INFECTIONS—SPECIAL REFERENCE TO ARTHRITIS. C. BERENS, D. M. ANGEVINE, L. GUY and S. ROTHBARD, Am. J. Ophth. 21: 1315 (Dec.) 1938.

Following experiments on rabbits, the authors give the following summary:

"1. Acute or chronic inflammatory lesions have been produced in the eyes of rabbits by intravenous injections of relatively small doses of the following organisms: Streptococcus hemolyticus, Staphylococcus aureus, B. typhosus, B. coli, B. proteus, and B. abortus.

"2. Various strains of streptococci, when injected intravenously, have produced more microscopic lesions of the uveal tract than any other organisms used.

"3. Multiple injections of streptococci were considerably more effective in producing lesions in the eye than single injections.

"4. The inflammatory lesions that were produced in a group of intradermally immunized (sensitized) rabbits were more frequent and extensive than in intravenously immunized or normal rabbits.

"5. Few eye lesions were produced by means of foci of infection established in various sites.

"6. The incidence of eye lesions was no higher in rabbits in which arthritis occurred than in those with no demonstrable involvement of the joints."

W. S. REESE.

VARIATIONS OF THE DEXTROSE CONTENT OF THE AQUEOUS HUMOR IN RABBITS AFTER REMOVAL OF SUPERIOR CERVICAL GANGLION. P. MAMOLA, Rassegna ital. d'ottal. 5: 180 (March-April) 1936.

The author reports a series of experiments which were made on rabbits, consisting of the removal of the superior cervical ganglion and the measurement of the change in the dextrose in aqueous humor.

Mamola concludes that after the removal of the ganglion the dextrose content of the aqueous humor of the homolateral eye increases but returns to normal after eight or ten days. Hardly any change is noticed on the aqueous of the other eye.

A. PERZIA.

EXPERIMENTAL ARGYROSIS OF THE CORNEA. A. RUSSO, Rassegna ital. d'ottal. 6:72 (Jan.-Feb.) 1937.

The author has produced argyrosis after many years of research. He has been able to demonstrate that while the silver impregnation does not appear under normal conditions of the conjunctival sac, it can be obtained by first inducing a chronic inflammation of the conjunctiva by the instillation of a solution of 25 per cent chloroform in olive oil and then instilling solutions of silver.

The biochemical picture of argyrosis obtained this way is the same as that seen in the cornea of man after the use of certain medical silver preparation. Histologic examination shows that Descemet's membrane presents silver deposits in the form of a ring or as spicules.

A. PERZIA.

General

RARE OCULAR DISEASES CONNECTED WITH DISTURBANCES OF THE MENSTRUAL CYCLE: REPORT OF CASES. L. NÉMETH, Klin. Monatsbl. f. Augenh. 101:83 (July) 1938.

Disturbances of the function of the endocrine glands may cause a number of ocular diseases and symptoms. They may be observed at any time during the entire cycle of menstruation from menarche to menopause but are more often associated with amenorrhea or scarce menses than with increased menstruation. They may be periodic, corresponding with the menstrual cycle, or of a vasomotor nature characterized by a hemorrhagic or exudative manifestation. The most common ocular disturbances are edema of the lids, inflammation of the palpebral margins and hordeola, due to vasomotor causes. Acne may produce palpebral involvement during eczema of the lids, and enlarged blood vessels of the eyeball may occur during menstruation. In 1 case Németh observed a flare-up of a small nevus of the bulbar conjunctiva during each menstruation, and Horwath reported the case of a woman with oligomenorrhea who had lymphatic eruptions during the menses. This patient received hormones for the oligomenorrhea for a week prior to the date of her menstrual period, with prompt result. Several cases of episcleritis have been reported due to the same cause. Spasm of the retinal vessels as a result of oligomenorrhea was observed in a patient aged 48 at the time of the approach of menopause. It was coupled with sudden embolism of the central retinal artery. The spasm returned, causing complete cessation of the circulation of the retinal blood.

This patient's daughter, aged 21, was suffering from transient obscured vision during the menses, resulting probably from vascular spasm. The occurrence of the disturbances in the right eye of mother and daughter suggests constitutional inferiority. Increase or decided decrease of intraocular tension was observed regularly during the menses. The etiologic factor, decrease of the endocrine secretion, may be caused by underdevelopment, diminished function or climacteric cessation of the function.

K. L. STOLL.

General Diseases

TERTIARY SYPHILIS OF THE EYE: REPORT OF A CASE. F. W. MEYER, Klin. Monatsbl. f. Augenh. 101: 390 (Sept.) 1938.

Meyer reports a case of tertiary syphilis in a woman aged 39 who had three allegedly healthy children. She was treated for chronic tuberculous scleritis and iridokeratitis. The clinical detail was typical for ocular tuberculosis without pulmonary involvement. The Wassermann reaction was negative at first, but was strongly positive when repeated after corneal bullae had developed. The initial syphilitic lesion occurred about ten to fifteen years previously. Blood transfusions were administered after the usual therapy failed. Temporary improvement followed, but after the third transfusion abdominal pain and severe headaches developed, then collapse and death followed. Autopsy revealed severe syphilitic mesaortitis and thrombosis of the carotid artery. The thrombus, which was found to be caused by an embolism of the middle meningeal artery, may have been dislodged by the transfusion, causing a cerebral hemorrhage. The histologic changes in the eye are described; they included edema of the optic nerve and retina, proliferation of the intima of the small blood vessels, increase of the glial cells and infiltration with round cells. The histologic changes of the optic nerve explain the concentric contraction of the field of vision.

K. L. STOLL.

Glaucoma

GLAUCOMATOUS DEGENERATION OF THE CORNEA. M. SALZMANN, Arch. f. Ophth. 139: 413 (Nov.) 1938.

According to Salzmann, much has been written about the changes which the cornea undergoes in glaucoma, and nothing essentially new can be added to this literature, especially since E. Fuchs has dealt extensively with these changes. With regard to details, the descriptions can be completed and existing conceptions can be more clearly and sharply defined. This Salzmann has done in 52 pages of text and with 37 excellent black and white drawings made from microscopic slides. The various glaucomatous changes in the cornea are classified under five headings: (1) changes of the epithelium, (2) changes of the superficial nerves, (3) glaucomatous pannus, (4) bullous keratitis and (5) the secondary forms of keratitis.

One often reads or hears that the clinically visible, transient glaucomatous opacity of the cornea disappears as soon as the intraocular pressure becomes normal. If such an opacity exists, it cannot be caused by any of the known histologic corneal changes, because these, or rather the reversible changes, disappear only slowly and gradually.

The anatomically visible changes of the epithelium are manifold (turgescence, shrinkage, necrosis and vacuolation). Vacuoles may develop within or between epithelial cells—a distinction which, histologically, is often difficult to make.

The best known and probably the most characteristic glaucomatous change occurring in the cornea is the abnormal visibility of the canaliculi of the rami perforantes in Bowman's membrane. In cases of glaucoma

these canaliculi are wider and contain a more eosinophilic substance; cells are seen growing through them toward the epithelium, at the base of which these cells may lead to the lamellar deposits, first described by E. Fuchs. Salzmann suggests the name neurodystrophia corneae superficialis (glaucomatosa) for the whole process.

Pannus should not be defined as granulation tissue, because the latter is soon transformed into tough scar tissue, whereas true pannus retains its loose texture for a long time. In contradistinction to the glaucomatous neurodystrophy, the glaucomatous pannus never destroys Bowman's membrane. Because of numerous instructive illustrations, this article, even without the text matter, makes good "reading."

P. C. KRONFELD.

CUSHING'S DISEASE COMPLICATED WITH GLAUCOMA: REPORT OF A CASE. J. TATÁR, Arch. f. Ophth. **139**: 793 (Dec.) 1938.

A Hungarian woman 24 years of age showed the characteristic symptoms of Cushing's disease (obesity, distention striae on the abdomen, hypertrichosis, polydipsia, polyuria and vascular hypertension). The entrance to the sella turcica and the sella itself were small; the dorsum sellae was thickened. There was slight exophthalmos. The eyes showed no signs of inflammation or of vascular decompensation. The anterior chambers were deep; the fundi and the central vision were normal. The intraocular tension varied during the short period of observation between 20 and 40 mm. of mercury. Miotics failed to keep the tension within normal limits. The visual fields were found to be concentrically constricted, perhaps because of spasms of the retinal arteries. The author speculates on the nature of the glaucomogenic factor in Cushing's disease.

P. C. KRONFELD.

Injuries

EPITHELIUM IN THE ANTERIOR CHAMBER OF THE EYE AFTER OPERATION AND INJURY. C. A. PERERA, Am. J. Ophth. **21**: 605 (June) 1938.

Perera gives a brief review of the literature on the downgrowth of epithelium into the anterior chamber. Following experiments on 8 rabbits, he gives the following summary and conclusion:

"1. Epithelial invasion of the anterior chamber of the human eye following operation or injury results in pearl tumors of the iris, epithelial cysts of the iris, or epithelization of the anterior chamber. This group of complications is due to implantation of epithelial cells within the anterior chamber or to growth of these cells through an operative or traumatic wound.

"2. The treatment of epithelial invasion of the anterior chamber has been unsatisfactory until recent years, when the use of radiotherapy has given encouraging results. A case of post-operative cyst of the iris following cataract extraction successfully treated by this method is reported by the author.

"3. A study of the literature on this subject, including reports of cases and experimental investigations, as well as examination of the pathological material in the collection of the Institute of Ophthalmology of the Presbyterian Hospital, New York City, shows that incarceration of iris or lens capsule following operation for removal of cataract is an important factor in epithelization of the anterior chamber. Hypotony plays an important role, appearing to favor growth of epithelium within the anterior chamber. Operative procedures which reduce the possibility of incarceration of iris and lens capsule will reduce the incidence of postoperative growth of epithelium in the anterior chamber of the eye.

"4. The author describes a study of the outcome of the experimental introduction of a flap of superficial corneal tissue into the anterior chamber of rabbits. The corneal epithelium, which proliferated rapidly immediately after the operation, gradually became degenerated and disappeared. The entire implanted corneal flap became absorbed within three weeks, with formation of an anterior adhesion of the iris in all instances. Epithelization of the anterior chamber of normal rabbits could not be produced by this method."

W. S. REESE.

Lacrimal Apparatus

INTRANASAL DACRYOCYSTORHINOSTOMY. P. PATRIARCA and C. DUC, Rassegna ital. d'ottal. 5: 205 (March-April) 1936.

The authors report the results of an intranasal operation on the tear sac of 10 patients affected with various forms of dacrocystitis: Rapid and complete cures were obtained for all, including 2 with phlegmonous dacrocystitis and a fistula.

The external method of dacrocystorhinostomy gave just as good results, and the operation is easier, since the bony wall can be trephined.

In each case the authors used a scalpel in making the opening in the bone. In many cases the trephine cannot be used because the nasal chamber is small and narrow.

A. PERZIA.

PLASMOMA OF THE LACRIMAL SAC. D. G. CARDELLO, Rassegna ital. d'ottal. 5: 239 (March-April) 1936.

The author describes 3 cases of plasma-cellular formation found in a series of 55 lacrimal sacs extirpated because of dacrocystitis. In all of the sacs there were rich nests of plasma cells in the inflammatory infiltration. The author concludes that these 3 cases are instances of pure inflammatory hyperplasia.

A. PERZIA.

Lens

THE PHYSICOCHEMICAL PROBLEM OF OPACIFICATION OF THE LENS. J. NORDMANN and P. REISS, Docum. ophth. 1: 461, 1938.

The literature on the electric potential of normal and cataractous lenses in relation to oxidation-reduction and its regulatory mechanism is reviewed. Various types of cataracts are considered.

The authors found that when lens mash was immersed in solutions of a decreasing p_H the clouding was more intense as the isoelectric point was approached. The effect of alkaline solution was less marked. The addition of oxidizing agents to a solution brought to the p_H of normal lens caused only a slight clouding. In slightly acidified solutions the same addition caused a clouding of the lens material which was considerable at p_H 7.0 under these conditions. The maximum clouding occurred at p_H 6.5, whereas without the addition of oxidizing agents it was necessary to go as low as p_H 5.0 to obtain quantitative precipitations. Whole lenses behaved similarly and became opaque at a potential of + 200 millivolts, the reaction becoming more intense when the p_H was slowly lowered.

By a combination of elevation of potential and of slight acidification, the authors were able to produce cataracts in vitro having physicochemical properties not unlike cataracts seen in vivo. The authors believe that this synergism occurs in naphthalene cataracts at the onset, in certain endocrine cataracts and in certain senile cataracts, at least during the evolution. It may apparently be admitted that for these forms of cataracts the theory of acidification is valid, providing the factor of the p_H is always taken into consideration. A slight lowering of the p_H in the absence of an elevated oxidation-reduction potential cannot explain a cataract. The authors reason that apparently variations of the p_H and of the oxidation-reduction potential are related, and they attach equal importance to the two factors. They conclude that ascorbic acid stabilizes the potential of the normal lens. It is not certain whether this mechanism holds for all types of cataracts.

E. G. SMITH.

Methods of Examination

SUBJECTIVE TESTS OF THE MUSCLE BALANCE, WITH A DESCRIPTION OF A SIMPLE AND PRACTICAL METHOD. A. BUCALOSSI, Ann. di ottal. e clin. ocul. **66**: 740 (Oct.) 1938.

The author's method of testing muscle balance depends on the suppression of fusion by means of a gray wedge placed before one eye. Sufficient reduction in the luminosity of the image seen through the wedge creates enough dissimilarity between the images of the two eyes to cause diplopia in persons with heterophoria, which is measured by the degrees of prism required to correct it. When a line of light is employed, absence of parallelism between the two images reveals cyclophoria. The degree of phoria may be read directly on a tangent scale instead of being measured with prisms.

S. R. GIFFORD.

Neurology

MARGINS OF THE OPTICALLY EXCITABLE CORTEX IN THE RABBIT. J. L. O'LEARY and G. H. BISHOP, Arch. Neurol. & Psychiat. **40**: 482 (Sept.) 1938.

The extent of the optically active cortex of rabbits was studied by recording active currents from the visual cortex after stimulation of the

optic nerve and by histologic methods after sections of the nerve. Results of the work show the optic area to be slightly larger than previously reported.

R. IRVINE.

EXPERIMENTAL STUDY OF MACULAR REPRESENTATION IN THE MONKEY. G. L. MAISON, P. SETTLAGE and W. F. GRETHER, Arch. Neurol. & Psychiat. **40**: 981 (Nov.) 1938.

Two theories explain the difference between the macula-sparing and the macula-splitting hemianopias found in man after interruption of the optic radiation. One theory holds that certain macular fibers in each optic radiation pass or send collateral branches, which pass through the splenium of the corpus callosum to the contralateral occipital cortex. According to this view, a lesion interrupting the optic radiation before these decussating fibers leave it results in macula-splitting hemianopia, whereas a lesion posterior to that point results in macula-sparing hemianopia.

The other theory interprets the clinical findings according to the postulate that the macula is diffusely represented throughout in the striate cortex. Thus, a lesion in the temporal lobe may result in macula-splitting hemianopia because all macular fibers in the optic radiation on one side have been interrupted. On the other hand, the usual occipital lobectomy results in macular sparing because, according to this theory, the most anterior portion of the striate cortex has been left intact.

The authors investigated the first theory by sectioning the optic tract on one side and removing the contralateral lobe in monkeys. No cortical vision should be observed in the preparation unless macular fibers or their collateral branches cross to the opposite occipital cortex. If a remnant of cortical vision existed, the theoretic pathway of impulses would be through the uncut optic tract, through the splenium and thence to the remaining occipital lobe.

Their results show no cortical vision existing after the operation, which is inconsistent with the first theory.

R. IRVINE.

THE ANATOMICOPATHOLOGIC AND PATHOGENIC CONDITIONS OF CEREBRAL SOFTENING. J. LHERMITTE, Bull. Soc. d'opht. de Paris **50**: 502 (Nov.) 1938.

The old conception of the production of cerebral softening has seen a change in the light of recent investigations. Necrosis of the nerve tissue due to the embolic phenomenon as described by Virchow and to thrombosis has been for the past the usual conception of the neurologist of the cause of cerebral softening. Recently Schaeffer and the author have studied the production of aseptic inflammation in relation to encephalitis and encephalomalacia. The discovery by Charcot and Bouchard of softening due to the formation of miliary aneurysm must not be neglected. Modifications in the cerebral circulation are often demonstrated by the fundic vessels as noted with the ophthalmoscope. Lhermitte stresses the relation of sympathetic and parasympathetic vascular reactions and their importance in vascular spasm, which may cause softening. Experimental work on animals with Schaeffer is described

and commented on. The conclusions show the various mechanisms which may influence the blood supply to the brain and reiterate that embolus and thrombosis are only two unusual causes for the condition.

L. L. MAYER.

PARTIAL HOMONYMOUS CHOKED DISK WITH PARTIAL ATROPHY OF THE OPTIC NERVES FOLLOWING A UNILATERAL LESION OF THE OPTIC TRACT: REPORT OF A CASE. O. FREUSBERG, Klin. Monatsbl. f. Augenh. **101:** 494 (Oct.) 1938.

A case is reported of a man aged 30 who suffered from headaches, vomiting and epileptiform attacks for ten years. Choked disks were noted, the condition being more marked in the right eye than in the left. The blindspot was slightly enlarged. Craniotomy after the method of Kümmel revealed a malignant small-celled glioma, the size of a child's fist, on the frontoparietal lobe. The growth was removed, but the trephining opening was not closed by a bony cover. Spastic hemiparesis and hemianesthesia of the left side followed, and the choking of the disks changed to slight atrophy with preservation of good vision for several years. Later headaches, epileptic attacks and other symptoms returned, a cerebral prolapse developed and psychic defects were observed. Rotary nystagmus, hemianopic pupillary reaction and left-sided homonymous hemianopia were some of the prelethal symptoms. Among other intraocular changes, the temporal half of the right disk was atrophic and the nasal half choked, the swelling amounting to about 4 diopters. No difference existed in the two halves of the left disk. Histologic examination of the optic nerve was done.

The tumor of the brain had destroyed the primary visual centers of the right side. This process caused partial degeneration of the peripheral visual tracts, causing the left-sided homonymous hemianopia. Later the increased intracranial pressure produced homonymous choked disk only in the area of the intact nerve fibers, which terminated in the intact left lateral geniculate body.

K. L. STOLL.

Ocular Muscles

STRABISMUS WITH PERMANENT POSTOPERATIVE DIPLOPIA. E. ADROGUE and J. J. LAGOS, Arch. de oftal. de Buenos Aires **13:** 512 (Oct.) 1938.

The case here reported was orginally one of convergent squint of a left amblyopic eye of 40 degrees which was operated on in 1934 by tenotomy of the internus muscle and advancement of the external rectus muscle.

In an examination of the "optic spacial sense" with the screen, a divergent strabismus of 14 degrees with 1.5 degrees of hyperphoria was found.

Examination of the diplopia showed it to be crossed on fixation of a 10 mm. object at 2 meters within a circle 3 to 4 cm. in diameter, the "crossed false image" being 23 degrees to the right and 4 degrees above the fixation point. A corresponding result was obtained with the Maddox rod test. On fixation with the amblyopic eye, the crossed

diplopia was of 12 degrees without vertical displacement. On examination of the "after-images" a crossed sensorial correspondence was determined. With the Tschermak test, monocular diplopia could be produced.

The authors conclude that in this case there was a permanent persistence of an abnormal correspondence, which after operation is usually temporary.

C. E. FINLAY.

Operations

TRANSPLANTATION OF THE CONJUNCTIVA OF THE CADAVER. M. ROSENTZWEIG, Vestnik oftal. **14**: 26, 1938.

For the purpose of transplantation, the author conserves the eye of a cadaver in 200 cc. of the cadaver's blood at a temperature of + 4 to + 6 C. without the addition of citrates. In 3 instances the blood was tested and was found to be sterile after nine days of conservation. The technic of the operation is given in detail.

Twenty-five transplantations of the conjunctivas of cadavers were done on 21 eyes for restoration of the conjunctival sac, for repair after removal of pseudopterygium, for pannus and for burns of the eye. The use of the cadaver's conjunctiva is particularly recommended for the treatment of burns of the eye, as the late results from the use of the mucous membrane of the lip are often not favorable and the patient is not subjected to any additional trauma. In 4 cases the transplant did not take, namely, in cases of complete ankyloblepharon and of severe pannus with entropion. In 2 cases of pannus the transplant had a therapeutic effect, as the pannus was liquidated and the cornea cleared markedly.

The length of the time of conservation of the eye was from one to twelve days. The difference in the type of the blood and the sex of the host and donor had no effect on the taking of the tissue. The time of observation was from a few weeks to one year. The blood of the cadaver is a good medium for conservation of the material.

Detailed histories of the patients and a table illustrate the article.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

INFLUENCE OF ENUCLEATION ON THE DEVELOPMENT OF THE CANAL OF THE OPTIC NERVE. E. LUZCA, Klin. Monatsbl. f. Augenh. **101**: 413 (Sept.) 1938.

Numerous recent publications show that changes of the canal of the optic nerve can be demonstrated in the living subject only roentgenographically. Unilateral pathologic changes yield the best result. The roentgenogram is indispensable in the diagnosis of atrophy and retrobulbar inflammation of the optic nerve and of papilledema; it is of importance in clearing up the cause of exophthalmos and in locating retrobulbar tumors and the extent of their growth. Differences between the optic canals of the two sides may exist physiologically according to

Goalwin. Nevertheless, the difference between the canals of the optic nerves in the roentgenogram may be the only means in arriving at a diagnosis. Luzca examined 37 patients who had had one eye enucleated. He found no difference in 18, 4 of whom had lost their one eye prior to their eighteenth year of life, whereas the remaining 14 had lost it after their twentieth year. The enucleation produced no differences in the form or measurement of the canals of the optic nerves at this age. Smaller measurements were obtained on the side of the enucleated eye for 15 patients who had had an eye enucleated at ages ranging from 3 to 20. One of these patients showed an enlargement of the canal of the optic nerve collateral with the enucleated eye. The causes for narrowing of the canal are atrophy or partial absorption of the dissected optic nerve, the physiologic stimulus of the growing optic nerve being terminated by the dissection. The development of the canal ceases, as a rule, in the eighteenth year of life. Comparison of the canals of the two eyes in the roentgenogram shows narrowing on the side of the enucleated eye.

K. L. STOLL.

Physiologic Optics

THE OPTICOPHYSIOLOGIC SIGNIFICANCE OF THE CENTERS OF ROTATION OF THE EYES AND THE DETERMINATION OF THEIR DISTANCE (BASE LINE). V. SPADAVECCHIA, Ann. di ottal. e clin. ocul. **66**: 681 (Sept.) 1938.

After reviewing the literature on this subject, the author admits that the center of ocular rotation varies in the living eye and that its determination must be only approximate or relative. The same is true of the base line, or the distance between the two centers of rotation. The author describes an apparatus by which he determines the corneal vertex of each eye when both eyes are fixing a target at distances varying from 30 cm. to 1.5 meters. From these data he calculates the base line. Determinations were made for 41 intelligent subjects. According to these determinations, the base line varied from a minimum of 53.6 mm. for women and 55.64 mm. for men to a maximum of 63.98 mm. for women and 67.17 mm. for men. Variations in the base line of the same person as measured for infinity and for 1.5 meters were noted, amounting to from 0.34 to 3.40 mm. Differences between the base line and the interpupillary distance were noted for all subjects, the greatest difference being 4.33 mm. This difference may be of importance in the centering of lenses when this is based on the interpupillary distance.

A bibliography accompanies the article.

S. R. GIFFORD.

Physiology

THE RESOLVING POWER OF THE HUMAN RETINA. F. DE LEONIBUS, Ann. di ottal. e clin. ocul. **66**: 750 (Oct.) 1938.

It is known that the resolving power of the retina is superior to that of the eye because of defects in the optic system. The author employed the method of Bruscaglioni, by which a diffraction grating

illuminated by monochromatic light is projected on the retina. The yellow portion of the spectrum, or the sodium light, was employed as a source of light. Detection of the diffraction lines was compared with detection of black and white lines, the latter representing the resolving power of the eye as a whole. The resolving power of the retina was always superior to that of the eye. Observations on 18 subjects showed it to correspond on the average to an angle of 26 seconds. The minimum corresponded to an angle of 17 seconds and the maximum to an angle of 50 seconds.

S. R. GIFFORD.

PROCESSES OF ADAPTATION IN THE VERTEBRATE RETINA IN THE LIGHT OF RECENT PHOTOCHEMICAL AND ELECTROPHYSIOLOGIC RESEARCH. R. GRANIT, Docum ophth. 1:7, 1938.

A comprehensive review of the more recent work on dark adaptation from the standpoint of photochemical and electrophysical research is presented. Some of the earlier work and theories are also briefly mentioned. The two major substances under consideration are visual purple and riboflavin. The variations in the reactions of the visual purple due to light, heat, p_H, time element and other factors are summarized, and the possible intermediate and end products of the reactions are mentioned. The more recent work on riboflavin is also discussed in relation to the subject, and several possibilities as to the significance of its presence in the retina are suggested.

The recent work on action currents in optic nerve fibers is also outlined. The various factors influencing the components of the retinograph and their possible significance are delineated.

The article contains a list of recent summaries and an excellent bibliography.

E. G. SMITH.

Retina and Optic Nerve

EXUDATIVE RETINITIS. H. D. LAMB, Am. J. Ophth. 21:618 (June) 1938.

Lamb refers to Coats and Leber's work on exudative retinitis and reports 8 cases giving the clinical and pathologic observation, from which he makes the following conclusions:

"Each of the eight eyes has the essential, primary change of infiltration with albuminous and edematous fluids in the whole or part of the retina. All layers of the retina are affected about equally in this infiltration, so that the process is diffuse and not external as Coats found. Two of the early cases show perivasculitis and macrophages on the inner surface of the retina, signifying an active inflammation in the retina. The other of the six early and the two of the late cases have a thin layer of connective tissue on the inner surface of the retina. Altogether six of the eight eyes have or have had an inflammation in the retina. In the remaining two early cases (2099 and 263), the inflammation had not attained sufficient intensity to develop macrophages. It is the macrophages, through their ability to become fibroblasts, that form the connective-tissue membrane on the inner surface of the retina. Therefore the essential process is a retinitis. The fluids infiltrating the retina

always appear under the retina as an exudate and cause a complete or partial detachment of the retina. Hence the designation exudative retinitis of Coats is accurate.

"The etiology of exudative retinitis is entirely obscure. The partial lipodystrophy in case 1398, the hypoövarianism in the mother of the patient in case 081, and the deficiency in the anterior pituitary in case 610 may be of some slight significance in pointing to the endocrine glands.

"Much of the most important secondary change in exudative retinitis is the reaction of the retinal pigmented epithelium to the inflammatory fluids within and under the retina. The pigmented epithelium proliferates and new pigmented cells arise internal to the parent layer. These proliferated cells are loose units with the ability to wander through the sub-retinal fluid and into the retina. These cells are therefore unquestionably macrophages in all situations except when in the process of proliferating. It is possible for pigmented epithelial cells to form elongated linear processes made up of closely coherent elements. Nevertheless, the pigmented epithelial cells probably always become macrophages before changing to fibroblasts. When fibroblasts are once developed, they possess to a high degree in themselves the faculty of proliferating. Connective tissue from pigmented epithelial cells of the retina developed in six out of the eight eyes of this series in four different positions: internal to the retina in a small mass in case 1460; within the retina in cases 2183 and 1325 in small amounts, and in case 610 from small to very large foci; at and near the ora serrata in cases 1398, 081, and 610, in small masses having a continuity with the retinal pigmented epithelium; and between the retina and the choroid in cases 081 and 610 in thick flattened masses.

"To the clinician, these anatomic findings are important, for out of the eight eyes, in only one, where detachment of the retina was not present at the time the eye was first examined, the suspicion of intraocular neoplasm could be avoided. In three eyes, increased intraocular tension accompanied the detachment of the retina." W. S. REESE.

ETIOLOGY OF IDIOPATHIC RETINAL DETACHMENT. A. E. MACDONALD, Am. J. Ophth. **21**: 658 (June) 1938.

MacDonald cites several cases in which retinal detachment developed following a severe lifting strain. He suggests an explanation based on the hydrodynamics of blood pressure in relation to cerebrospinal fluid pressure. W. S. REESE.

INFLUENCE OF MARCONITHERAPY (SHORT WAVE THERAPY) ON RETINITIS PIGMENTOSA. A. BUCALOSSI, Ann. di ottal. e clin. ocul. **66**: 641 (Sept.) 1938.

The literature on the pathogenesis and treatment of retinitis pigmentosa is reviewed. The author employed short wave therapy in 7 cases of this disease in the hope of favorably affecting the capillaries and the peripheral circulation. An Italian apparatus was employed which had a potential of 320 watts and delivered wavelengths of 6

meters. A glass-covered Schliephake electrode, 3.5 cm. in diameter, was placed 1.5 to 2 cm. from the eye, the indifferent electrode being placed on the neck. A current of 18 to 21 volts was employed for eight to twenty minutes. Treatments were given in series of ten on alternate days to each eye. Visual acuity was improved in each eye in 5 of the 7 cases and in one eye in a sixth case. The visual fields showed improvement in 4 cases. In 1 case in the early stage a partial ring scotoma disappeared entirely. The dark adaptation in the 3 cases in which it was determined showed a definite lowering of the luminous threshold. Dilatation of the retinal vessels was noted in several cases, while in others it could not be noted, in spite of functional improvement. Functional improvement was noted in most cases after the first or second treatment and was maintained during one to eight months of observation. In 1 case a slight decrease of function occurred one month after suspension of treatment, but this loss was recovered after a second series of five treatments. Best results were obtained in the cases in which the condition was not far advanced; in 1 case in which it was advanced there was no improvement.

A bibliography is included.

S. R. GIFFORD.

PERFORATION OF THE MACULA BY INDIRECT TRAUMA. A. BUCALOSSI, Ann. di ottal. e clin. ocul. **66**: 768 (Oct.) 1938.

A case is reported in which a traumatic macular hole was found after a severe blow on the eye. The retina below the hole was also involved, resulting in loss of the upper half of the field and secondary atrophy of the optic nerve.

The literature is reviewed, and a bibliography is included.

S. R. GIFFORD.

VITAMIN B_1 IN THE TREATMENT OF RETROBULBAR NEURITIS. V. PATERNOSTRO, Ann. di ottal. e clin. ocul. **66**: 891 (Oct.) 1938.

Crystalline vitamin B_1 was administered in 20 cases of retrobulbar neuritis due to varying causes. Two milligrams of the product employed contained 1,000 international units. In 8 cases the condition could be classed as tobacco-alcohol amblyopia. Marked and rapid improvement in vision occurred in all 8. In 3 cases vision returned to normal within a week. From six to nine daily intravenous injections, usually of 5,000 international units, were given. This was followed by the ingestion of a diet rich in vitamin B_1 with, as a rule, the administration of some form of vitamin concentrate by mouth. In 7 cases the visual improvement remained after such treatment, the time of observation being a year in 1 case. In 1 case the vision, after having risen from 1/10 to 7/10, dropped to 2/10 soon after treatment was stopped and was not improved by further treatment. In 4 cases the retrobulbar neuritis was considered due to paranasal sinusitis. Results were negative in 3 of these cases, but apparently positive in the fourth. Two of the patients had diabetes, 1 of whom also overindulged in the use of alcohol and tobacco. Each patient obtained good results. In 2 cases in which the neuritis was considered of rheumatic origin the results of treatment were

good. Some improvement was noted in 1 case of syphilitic origin and in 1 considered to be due to gout. In 2 cases in which the etiologic agent was unknown treatment was without benefit.

A bibliography accompanies the article.

S. R. GIFFORD.

Trachoma

THE EFFECT OF TARTAR EMETIC ON THE COURSE OF TRACHOMA. L. A. JULIANELLE, R. SORY, J. E. SMITH and A. C. LANGE, Am. J. Ophth. **21**: 651 (June) 1938.

Following the treatment of patients with various types of trachoma, the authors give the following summary:

"A technique is described for the treatment of trachoma with tartar emetic [antimony and potassium tartrate]. The indications are that in certain varieties of this disease this drug may be an effective therapeutic agent.

"The various manifestations most likely of successful treatment have been discussed in brief, and subsequent study will determine the true value of the impression expressed at the present time."

W. S. REESE.

THE SUMMER ANTITRACHOMATOUS COLONY AT MONDELLO. D. D'AMICO, Ann. di ottal. e clin. ocul. **66**: 214 (March) 1938.

The activities of this colony for children with trachoma near Palermo, Italy, are described. They include not only local treatment but general physical care, especially organized outdoor exercise.

S. R. GIFFORD.

IMPORTANCE OF THE WEIL-FELIX REACTION IN TRACHOMA: REPORT OF CASES. S. POŠTIĆ, Klin. Monatsbl. f. Augenh. **101**: 341 (Sept.) 1938.

Poštić discusses in detail the research on the causation of trachoma and the tests employed. He used the Weil-Felix reaction for patients with different stages of trachoma, accepting MacCallan's classification of four stages. The results are recorded in a number of tables. To control the results of the Weil-Felix test for trachomatous patients Poštić employed the test on patients suffering from ocular diseases other than trachoma and on a group of patients free from any pathologic ocular symptoms. The Wassermann reaction in syphilis is similar to the Weil-Felix reaction in trachoma as it is especially positive at the height of the disease. This experience would point toward a certain parallelism between these two diseases, a fact which is supported by the recent view that the rickettsias are protozoa. The Weil-Felix test was positive for 37.5 per cent of patients with the first stage of trachoma, for 80 per cent with the second stage, for 71.9 per cent with the third stage and for only 60 per cent with the fourth stage. Not counting possible interferences with the result by former spotted fever, the Weil-Felix reaction is indisputably of importance in demonstrating the role played in trachoma by the rickettsias.

K. L. STOLL.

Tumors

METASTATIC CARCINOMA OF THE IRIS. T. E. SANDERS, Am. J. Ophth. 21: 646 (June) 1938.

Sanders remarks on the rarity of metastatic carcinoma of the iris and reports a case. He gives the following summary:

"The literature of metastatic carcinoma of the iris is reviewed.

"The fourth case of metastatic carcinoma of the iris without other uveal involvement is reported.

"The histologic findings in the first case in which the intact globe was examined are described.

"The origin, distribution, and clinical findings are discussed."

W. S. REESE.

SARCOMA OF THE IRIS. H. D. ROSENBAUM, Am. J. Ophth. 21: 1360 (Dec.) 1938.

Rosenbaum reports the case of a 26 year old woman who had had a yellowish brown spot on the iris all her life which began to increase in size during the last three years, until it finally invaded the pupillary area. The eye was quiet and painless except for an occasional slight ache and vision of 6/7.5. It was enucleated, and a glass ball was implanted. The pathologic report is given, and the following comment was made:

"According to Callender and Wilder, the tumors containing epithelioid, fascicular, and mixed-cell types are more malignant than those in which the predominating cell is the spindle-cell. This is borne out in this case; here the constituent cell was one containing nuclei, having spindle shapes of varying lengths and thicknesses. Another factor used to determine the relative malignancy of this type of growth is the presence and location of the argyrophil fibers. The above authors concluded that the presence of these fibers in all areas of the primary tumor is indicative of comparative benignancy; however, those that contained no fibers in the primary tumor were of the more malignant class. The reticular cells in the case reported were few in number, but were located around the tumor cells."

W. S. REESE.

PRIMARY EPITHELIOMA AND CARCINOMATOUS EPITHELIOMA OF THE LIMBUS. E. BUSSOLA, Rassegna ital. d'ottal. 5: 434 (July-Aug.) 1936.

The author describes a case of primary epithelioma of the limbus which secondarily extended over the cornea and conjunctiva.

The histologic picture at first observation was similar to the clinical and the histologic picture of the group of tumors classified by Poleff as "initial epithelioma," while at the second observation the picture seemed to belong to the group of epithelioma carcinomatosis. The histologic picture presented some of the characteristics of a malignant growth.

A. PERZIA.

Uvea

DIAGNOSIS OF BOECK'S IRITIS. BLEGVAD, Acta ophth. **16**: 598, 1938.

The author agrees with Osterberg's conclusion that there is a particular form of iritis which may occur in cases of Boeck's sarcoid and which is not identical with tuberculous iritis. Boeck's iritis is less painful and less destructive and gives rise to less proliferative change than does tuberculosis. Blegvad differs from Osterberg, however, in that he believes it often possible clinically to distinguish between the two conditions on morphologic grounds, especially if the iritis is nodular. In tuberculosis the iris nodules are dirty white with a faint yellowish tinge. They are almost always round or oval with a smooth surface. They come from the depths of the iris tissue and seem to push the iris fibers aside as they do so. The blood vessels pass over or around them. If the nodules become smaller, the smallest ones may leave behind almost no trace, but the larger ones leave areas of atrophy in the iris, which may therefore appear punched out. In Boeck's iritis the nodules are large, not smooth and round, but rough and irregular. They are reddish yellow and are woven through by a mass of small blood vessels which may branch superficially. The nodules seem to be of firm consistence, and if they reach the angle of the anterior chamber and push against the posterior surface of the cornea they may bend the iris backward or even grow through the sclera. The general tendency, however, is toward healing. The nodules gradually become smaller and finally disappear, leaving no trace of their presence in the iris tissue. O. P. PERKINS.

Vitreous

THE QUESTION OF THE VISIBILITY OF THE HYALOID CANAL. F. ANTINO, Ann. di ottal. e clin. ocul. **66**: 38 (Jan.) 1939.

The author prefers the term "visibility of the hyaloid canal" to "persistence of the hyaloid canal," since the canal is normally present in life but is not visible by ordinary examination. On the basis of the literature, he classifies the cases in which the hyaloid canal was visible during life as follows: (1) those in which the canal was visible as a tubular formation with more or less transparent walls and was not accompanied in its course by vessels; (2) those in which the canal was accompanied in its course by vascular formations; (3) those in which the lumen of the canal was completely or partially occupied by opaque formations distinct from the walls of the canal, and (4) those in which the canal was only partially visible. In addition, the literature contained observations on the so-called persistent hyaloid filament, which may be related to the visible hyaloid canal.

From the variability of the reported findings, especially as regards size and shape, the author believes that many reported cases concern persistence of the hyaloid canal as a congenital anomaly. He reviews the few reported cases of acquired visibility of the canal resulting from hemorrhage or inflammatory products and reports 2 unusual cases.

The first case was seen soon after an injury, with diffuse hemorrhage into the vitreous. Seven months later the vitreous had cleared, with return of vision to 4/10. An opaque cord was seen extending from the papilla through the vitreous to end at the nasal portion of the lens. The posterior end of the formation was enlarged to cover the disk,

while the anterior end also spread out to form a funnel-shaped attachment to the lens. Two months later the posterior portion of the cord had entirely absorbed. After six months there was no further change.

The second case was that of a girl of 12 with myopia of 34 diopters in the right eye and 27 diopters in the left eye. The left fundus showed marked depigmentation about the disk and a cord similar to that seen in the preceding case, extending from the disk to the posterior pole of the lens. There was no change in the picture during several months' observation. An area adjacent to the disk suggested the existence of a healed inflammatory lesion, and the author considers the opaque structure as probably resulting from the collection of inflammatory products in the hyaloid canal. He believes that some of the cases reported as congenital anomalies may have really represented similar processes.

A bibliography and three colored plates of the fundus accompany the article.

S. R. GIFFORD.

Therapeutics

INDUCED HYPERPYREXIA IN OPHTHALMOLOGY. W. S. WEEKS and S. A. MORRIS, Am. J. Ophth. 21: 664 (June) 1938.

The authors describe the production of, and contraindications for, hyperpyrexia effected by an inductotherm by which an electromagnetic field is produced with the patient acting as a resistance. They cite a number of cases and draw the following conclusions:

"It is obvious that one is not justified in drawing conclusions from so small a series of cases. However, it would appear that physical fever therapy is of value in the ocular complications of gonorrhea, in iritis, in superficial punctate keratitis, and in scleritis; but seems to be of little or no value in cases of trachoma, luetic uveitis and interstitial keratitis.

"The most striking evidence of the efficacy of fever therapy was observed in the cases of iritis, in which the rapid disappearance of gross exudate was noted during the treatment. This rapid absorption of exudate undoubtedly plays a significant role in preventing the formation of synechiae."

W. S. REESE.

TREATMENT OF OCULAR DISEASES BY THE ORBITAL INJECTION OF ALCOHOL. L. WEEKERS, Ann. d'ocul. 176: 81 (Feb.) 1939.

The practice of orbital injections of alcohol in ocular therapeutics is widespread, but this method still inspires fear in some areas. For about ten years now ophthalmologists have been using this form of treatment, and it would be difficult for them to stop.

In 1930 Weekers insisted that one could with impunity to the vision inject alcohol into the orbit, provided the concentration was not more than 40 per cent, and that by its use certain ocular conditions were cured. The reports of other writers are commented on, and a special note is made of the reports of Magitot.

The article is divided into sections dealing with the action of orbital injections of alcohol on ocular pain; the action on ocular tension and irritating symptoms, such as photophobia, and blepharospasm; the action on ocular lesions and their course; complications resulting from these injections, and recent views regarding the physiopathologic picture and the treatment of pain.

S. H. MCKEE.

EFFECT OF SHORT WAVE THERAPY ON THE RETINAL CIRCULATION. M. CORRADO, Ann. di ottal. e clin. ocul. 66: 721 (Oct.) 1938.

The author employed irradiation by short waves (30 meters) and ultra-short waves (6 meters) on the eyes of rabbits. Observations of the fundi were made at intervals of fifteen to thirty minutes after the treatment. Eyes were removed for histologic examination either immediately after treatment or twenty-four hours later. Treatments with the 30 meter instrument were divided into those of slight, medium and great intensity, the current for each group being 19, 21 and 22 volts, respectively. The smaller intensities resulted in inconstant dilatation of the vessels of the fundi and slight congestion of the choroidal vessels as seen in sections. In animals receiving the larger doses, these changes were constant and more marked, being still evident in eyes enucleated twenty-four hours after the largest dose was given. Animals receiving treatment with ultra-short waves showed similar changes, but they were constant and persisted for twenty-four hours, even with the smaller dose. No other changes in the tissue were observed. The congestion involved not only the arterioles and capillaries but the veins of the retina and the choroid. The evidence seems to indicate that doses of the smallest degree employed, 17½ volts for thirty minutes, utilizing ultra-short waves, is sufficient to produce the desirable vasodilatation without risk of producing undesirable changes in the tissue. S. R. GIFFORD.

AUTOHEMOTHERAPY AND HYPHEMIA. G. LECHERI, Rassegna ital. d'ottal. 6: 62 (Jan.-Feb.) 1937.

The author employed autohemotherapy in 9 cases of hyphemia following iridectomy and in cases of glaucoma hemorrhagica, intravitreous hemorrhage and traumatic or spontaneous hyphemia. The results in 3 cases were good; the hyphemia was rapidly absorbed. A. PERZIA.

CLINICAL OBSERVATIONS ON INJECTIONS OF BLOOD INTO THE ANTERIOR CHAMBER AFTER THE METHOD OF SCHIECK IN SOME DISEASES OF THE EYE. L. SATZ and N. MEDVEDIEV, Vestnik oftal. 14: 61, 1938.

Satz and Medvediev used autohemotherapy for 4 patients with tuberculosis of the eye, for 3 patients with severe trachoma, for 1 patient with corneal ulcer and for 1 with bilateral uveitis of indefinite causation. One patient received three injections, 3 patients received two injections and 5 patients received one injection. Local therapy was applied simultaneously. The technic of the operation was modified as follows: An opening is made in the cornea with a fine knife, and aqueous escapes; a dull rather heavy needle is inserted, and 0.1 cc. of blood is slowly injected around the limbus, the pupillary area often being left free of blood. In 2 tuberculous eyes there was marked improvement of vision; in 2 trachomatous patients the inflammatory symptoms subsided, and the vision was improved greatly. The blood absorbed within a week.

O. SITCHEVSKA.

Society Transactions

EDITED BY W. L. BENEDICT

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

S. JUDD BEACH, M.D., *Chairman*

Ninetieth Annual Session, St. Louis, May 15-19, 1939

DERRICK VAIL, M.D., *Secretary*

PROBLEMS IN SPECIAL STUDY FOR GENERAL PHYSICIANS: Chairman's Address. DR. S. JUDD BEACH, Portland, Me.

Largely through the influence of the American Board of Ophthalmology, training methods for ophthalmologists have been raised to a high degree of excellence. The number of opportunities is, however, totally inadequate.

The approved training for those who can get it is the complete integrated course. At its best it correlates formal study and didactic lectures with the clinical experience of a residence. It is designed to take the novice and after one or more years turn him out ready to undertake special practice.

In contrast to this there are what have been called "extension courses." These usually consist of one or two hours of rather elementary and often informal demonstrations. The time limitation holds such conferences to a restricted field. Their greatest value has probably been to bring to practitioners the newer points of view and discoveries. Even if there were enough places for all applicants, neither of these methods of teaching entirely meets the needs of the general practitioners who for whatever reason find themselves in ophthalmologic practice without adequate preparation. Many of them cannot spare the time for consecutive formal training. For these "forgotten men," four expedients have been discussed by those interested:

1. The so-called "progressive" courses should be made available in less favored parts of the country. These courses consist of conferences taking from three to four days to a fortnight. They are patterned after the outstanding one in ophthalmic pathology conducted annually by the American Academy of Ophthalmology and Otolaryngology. At various other instructional sessions different subjects have been handled in the same way with equal success. Courses in orthoptics and in practical surgery are regularly oversubscribed. They should be equipped with clinics and moving pictures and extended to cover the rest of the subjects.

2. A literature for self teaching, suited to home study, either with or without courses like those now being arranged for residents, should be devised. The lay institutions engaged in teaching by mail have long recognized that most textbooks for intramural classes are rather works of reference than works adapted to home study.

3. Suitable tests should be provided for checking progress made by students with either of these methods, adapted like the familiar achievement tests for self correcting or rating by clerks.

4. The manual and the examination should be combined in the same volume, as is done in the familiar self-teaching textbooks equipped with review questions and correct answers.

THOUGHTS ON OFFICE PRACTICE. DR. ALLEN GREENWOOD, Boston.

Having been in private practice for close to forty-nine years, I have had many interesting experiences. These have led me gradually to formulate some thoughts on what to do and what not to do in the handling of patients. An ophthalmologist who gains the confidence of his early patients will unquestionably over a period of time add to the number of patients who are recommended to him by those who have confidence in him.

Such confidence may be inspired in the patient by following three rules: First, be honest. Second, be thorough. Third, do not criticize the work that others have done prior to the patient's appearing. One should be willing to explain to the patient things about which he should really have a fair amount of understanding. One should not talk too much or promise too much. If the patient is wearing glasses which one feels after examination are not suitable, one should simply tell the patient that there has been a change in the eyes and that it is necessary to change the glasses correspondingly. One should not bluntly state that the glasses are all wrong and that the oculist has made a mistake. One should try to follow the Golden Rule in one's attitude toward the work of others and in all ways.

I have mentioned especially the desirability of thoroughness. Let me indicate what I consider a thorough examination to consist of: (1) observation of the patient and his eyes while taking a careful history; (2) a good examination of the front of the eyes with oblique illumination and loup or with the slit lamp, with particular attention to the appearance of the cornea, iris, pupil and lens; (3) the careful use of the retinoscope for quick determination of the gross refractive error, with or without a cycloplegic; (4) careful examination of the fundus, including the peripheral portions as well as the disks and maculas (I, personally, feel that this should never be omitted), and (5) the use of the case of trial lenses, with some of the recognized methods of working out the refraction in this way.

With the finding of the error of refraction, the muscle balance should be tested for far and near vision. It is my custom usually to try the fields by the confrontation method. This can be done quickly, and if no abnormality is found it is not necessary, ordinarily, to use the perimeter or the tangent screen.

Then comes the task of telling the patient what is necessary for him to do. Let me caution particularly against saying anything to the patient that will cause fear on his part. The only exception to this rule is in case a melanoma or definite glaucoma is found. Then it is sometimes necessary to speak with conviction and force in order to prevail on the patient to have whatever is necessary done.

Some time should be spent in telling the patient how he can most easily become accustomed to the wearing of glasses for the first time or how he can get used to a radical change. Particularly is this true of the patient who is getting bifocal lenses for the first time. It is wise to have the majority of patients return for a check-up on the refraction after they have worn their glasses for a while.

For patients who have conditions that require enucleation, it is a little better to lead up to the operation gradually, even when a malignant lesion is suspected.

DETECTING, MEASURING, PLOTTING AND INTERPRETING OCULAR DEVIATIONS. DR. WALTER B. LANCASTER, Boston.

This article was published in full in the November issue of the ARCHIVES, page 867.

THE TREATMENT OF SECONDARY GLAUCOMA. DR. SANFORD R. GIFFORD, Chicago.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

IRIDENCLEISIS IN GLAUCOMA. DR. EVERETT L. GOAR and DR. JACOB F. SCHULTZ, Houston, Texas.

This article is published in full, with discussion, in this issue of the ARCHIVES, page 1035.

THE VALUE OF EARLY OPERATION FOR CHRONIC PRIMARY GLAUCOMA. DR. ALGERNON B. REESE, New York.

For the purpose of this study there were selected 105 cases of chronic primary glaucoma in which an operation had been done with a trephine and in which the fields of vision and blindspots and the tonometric readings were recorded before the operation and repeatedly after it for an average of five years. These cases were divided arbitrarily into cases of early involvement and cases in the advanced stage. In over 90 per cent of the former the disease was arrested. Of the latter, progressive contraction of the visual fields and enlargement of the blindspot occurred in 72 per cent, in spite of the fact that in 60 per cent there was normal intraocular tension after the operation. The importance of early diagnosis and early operation when indicated is emphasized.

DISCUSSION

DR. HARRY S. GRADLE, Chicago: The conclusions that Dr. Reese has reached and so ably presented should be spread broadcast, not only for the purpose of curing the intolerable pessimism of many ophthalmologists regarding glaucoma but for the purpose of urging those with glaucoma to seek attention early.

The division of the cases into those in the early stage and those in the advanced stage is most proper. Parsons, who stated that every patient with glaucoma should be operated on as soon as a diagnosis was made, has changed his opinion and now considers operation indicated for persons with advanced glaucoma and for unreliable clinical patients.

There is no question that a case of advanced compensated glaucoma with the classic appearance of the cupped disk and the extensive changes in the visual field calls for immediate surgical treatment; however, many more cases of early involvement are being observed now than in the past, thanks to the better diagnostic methods.

I do not believe that Dr. Reese emphasized sufficiently the fact that when operation is indicated early intervention is desirable. It is true that if the disease cannot be controlled by other than surgical methods, temporizing is to be avoided; early operation will produce the results that Dr. Reese has shown, but there must be decided indications for surgical intervention. One is not justified in operating in cases of early compensated glaucoma until one is sure that the condition cannot be controlled by nonsurgical treatment. That takes time. One cannot be sure of the lack of control by nonsurgical methods until many weeks or even many months have elapsed. Neither can one be sure of lack of control if tonometric measure of intraocular pressure is depended on entirely. A given level of intraocular pressure may be obtained that can be held by miotics, but whether that intraocular pressure is pathologically high for the eye in question can be determined only when the effects on the visual field and the central visual acuity have been determined. These effects are slow in making their appearance, and one must not judge too hastily that an eye requires surgical treatment until one is sure that the intraocular pressure cannot be maintained medicinally. Therefore, I am trying to stress the necessity of strenuous indications for operation, but when it is indicated Dr. Reese is absolutely correct; nothing should be allowed to interfere with its immediate performance.

Dr. Algernon B. Reese, New York: I should like to mention one finding in the analysis of these cases which I thought was of interest and that is that 25 patients among those with advanced glaucoma showed progressive constriction of the visual field to the point of so-called telescopic fields and then showed no further progression of the condition over the follow-up period. Central field remained in 12 patients in spite of the fact that the intraocular pressure stayed above normal. Ten of the 25 patients retained special vision, and 5 of these showed increased intraocular pressure over the follow-up period.

The explanation for this seems to be that in some eyes the macular region received sufficient nutrition from the underlying choroid to enable it to function independently of the retinal blood supply or when altered by the glaucoma.

Cyclodialysis with Insertion of a Metal Implant in the Treatment of Glaucoma: A Preliminary Study. Dr. Manuel U. Troncoso, New York.

This article will be published in full, with discussion, in a later issue of the Archives.

Ocular Leprosy in the United States: A Study of 350 Cases. Dr. John J. Prendergast, St. Paul.

This article will be published in full, with discussion, in a later issue of the Archives.

PRIMARY TUMORS OF THE OPTIC NERVE: A PHENOMENON OF RECKLINGHAUSEN'S DISEASE; A CLINICAL AND PATHOLOGIC STUDY WITH A REPORT OF FIVE CASES AND A REVIEW OF THE LITERATURE. DR. FREDERICK A. DAVIS, Madison, Wis.

Primary tumors of the optic nerve are comparatively rare; about 380 cases are on record. Microscopic study reveals that most of them are gliomas or endotheliomas. The histogenesis of the neoplastic cells has not been fully determined. A study of the microscopic structure of 5 primary gliomas of the optic nerve associated with Recklinghausen's disease showed considerable variation. There were five distinct stages in the evolution of the growths. So-called primary tumors of the optic nerve are in many, perhaps most, instances but a part of a widely disseminated lesion of the central and peripheral nervous system commonly known as Recklinghausen's disease. Removal of the tumors by operation, with preservation of the globe, is advised by many authorities.

DISCUSSION

DR. PERCIVAL BAILEY, Chicago: Dr. Davis has made a most careful and useful study of these tumors, and his conclusions seem to me to be perfectly logical. These gliomatous tumors may involve the optic nerves in either their extracranial or their intracranial portion or both. In either case they may be associated with manifestations of Recklinghausen's disease elsewhere in the body. In 1930 Fulton and I published a report of such a glioma of the optic nerves (Nueva contribucion sobre los tumores del tercer ventriculo: Su associacion con el sindrome de Recklinghausen y con el edema de Quincke, *Arch. argent. de neurol.* **5:** 1-27, 1930). The patient, a girl of 6 years, had numerous pigmented areas in the skin over the buttocks.

When these gliomas occur intracranially they are usually composed of spindle-shaped cells (Bailey and Eisenhardt: *J. Comp. Neurol.* **56:** 391-430, 1932). Even Lundberg admits this but follows Hortega in maintaining that the neoplastic cells are of oligodendroglial origin. Russell and Bland believe them to be astrocytic. Whatever the exact nature of the intracranial tumors, Dr. Davis has shown that those which develop in the intraorbital portion of the optic nerves are composed mainly of astrocytic cells with a variable admixture of oligodendroglial cells.

His demonstration of the glial nature of the proliferation in the pia-arachnoid is important. Oberling has long maintained, on the basis of such extensions of glial tissue into the leptomeninges over the brain as well as over the optic nerves, that the leptomeninges are of neuroectodermal origin and has thereby caused great confusion in the terminology of intracranial tumors. It seems to me that Dr. Davis has settled the problem so far as the orbital tumors are concerned. The glial proliferation in the leptomeninges is of optic origin, and the arachnoidal proliferation is secondary.

The operative approach which he has used is logical and safe and effectively answers Dandy's dogmatic disapproval of an ophthalmic approach. No one who knows how little can be accomplished by intracranial operation could be very enthusiastic for the transfrontal route. In spite of the rarity of these tumors, Dr. Davis has rightly called the

attention of ophthalmologists to their association with other manifestations of Recklinghausen's disease, a lesson which might profit all physicians, especially neurologists and neurosurgeons.

DR. JESSE M. LEVITT, Brooklyn: Dr. Davis' monograph is a distinct contribution to the pathology of tumors of the optic nerve and for the most part confirms the views of Dr. Hudson and Dr. Verhoeff, to whom credit is due for the present conceptions. The pathogenesis of gliomas of the optic nerve has been clearly described and excellently illustrated and their intraneural and interstitial origin proved beyond any doubt.

The main thesis of the presentation has been a correlation of tumors of the optic nerve with neurofibromatosis, or Recklinghausen's disease. Owing to the extreme rarity of these tumors, this study of 5 is of great interest in itself. The coexistence with neurofibromatosis in each case is of special significance. In many of the cases of tumor of the optic nerve reported in the literature the presence or absence of neurofibromatosis has been noted, but that the relation between the two is more than incidental has only been suggested within the last few years. Both conditions are considered to be congenital anomalies in the development of the nervous system and may well have the same basic causation, whatever that may be, though they manifest themselves in apparently unrelated pathologic or microscopic appearance. In correlating peripheral with central neurofibromatosis (which include intracranial neoplasms of various pathologic types), Turner and Gardner (*Am. J. Cancer* 32: 338, 1938) recently suggested that Recklinghausen's disease be regarded as a disease process which involves all of the sheaths and enveloping membranes of the nervous system and that this concept, in a broad sense, be extended to include the glial elements as well. Dr. Davis has shown that about 10 per cent of the cases of tumor of the optic nerve reported in the literature (which includes the 5 cases in the present paper) were associated with peripheral neurofibromatosis. This percentage possibly is considerably higher, considering that the lesions of Recklinghausen's disease may have been overlooked or disregarded and the fact that 60 to 75 per cent of the cases of tumor of the optic nerve occur in the first decade of life, while the manifestations of peripheral neurofibromatosis usually become evident at puberty and in many cases later in life.

I wish to report briefly 3 cases illustrating the three different clinical types of tumor of the optic nerve: (1) intracranial, (2) intraorbital and (3) intraocular.

The first case, which I reported in the ARCHIVES (18: 91 [July] 1937), is that of a 43 year old white woman who had progressively lost vision in the left eye over the course of many years until she was completely blind and who had painless proptosis and ptosis of the same eye of recent origin. General physical examination and laboratory studies gave negative results; there was no neurofibromatosis. Craniotomy was performed, and a massive chiasmic growth was exposed. Removal was not attempted. The patient died on the twelfth postoperative day, and postmortem examination showed a glioma of a vascular nature.

The second case, reported through the courtesy of Dr. I. D. Kruskal, of Brooklyn, is that of a 9 year old white girl who had been entirely blind in the right eye for a year and a half and whose parents reported progressive proptosis of the same eye over the course of a half year. General examination gave negative results; there was no neurofibro-

matosis. Roentgenograms disclosed a generalized haze in the left orbit, interpreted as a soft tissue infiltration; the optic foramens and skull were normal. A course of irradiation therapy was without beneficial result. A Krönlein operation was performed, and a mass the size of a hazelnut, filling the apex of the orbit and surrounding the optic nerve, was exposed and removed. Dr. Bernard Samuels reported the growth to be a glioma of the optic nerve, probably an astrocytoma, and Dr. Verhoeff classified it as a coarsely reticulated type of glioma.

The third case is one of suspected neoplasm of the optic nerve head. The patient, a 46 year old white man, first examined in October 1934, has an immobile, solid appearing mass in the left eye which projects into the vitreous in front of the optic disk and completely obscures it. The surface of the mass is smooth and pinkish, owing to innumerable fine blood vessels. It is capped by an irregularly crescentic zone of blood red hue. Pressure on the globe blanches the surface, and on release of pressure the color becomes deep orange and then pinkish yellow. There are several small discrete white spots in the fundus temporally.

With the exception of slight displacement of the retinal vessels emerging from the nasal side of the disk zone, the vessels appear normal. The eye is outwardly normal; there is no proptosis. The tension is normal. The peripheral field of vision is full; the blindspot is markedly enlarged and almost reaches the fixation zone; the optic foramen is of normal size. The Wassermann reaction of the blood is negative. The vision of the eye is 20/200 with correction and has not altered during the four and a half years of observation. There has been a slight increase in the size of the mass and an appreciable increase in the number of white spots in the fundus. A cataract was removed from the other eye by the intracapsular method in May 1938, and the fundus is entirely normal. It is largely speculative as to the type of lesion that is present, but owing to the obvious vascularity it is felt that the tumor may be of vascular origin.

DR. F. PARK LEWIS, Buffalo: It may be worth while to mention briefly a case of this kind because of the fact brought out by Dr. Davis that the condition is of such infrequent occurrence. He reported 300 cases. I have no doubt that if cases that have come under individual observation were also reported one would find they are much more common than is believed.

A case of this character came under my observation about two years ago. There was marked proptosis in the right eye and atrophy of the optic nerve, which had been gradually progressing for five or six years. The proptosis measured about 4 mm.; there were no evidences of Recklinghausen's disease. This case was particularly interesting, because as I had a case of Recklinghausen's disease at the same time a comparison of the two conditions was easily made. There was no involvement anywhere except for the tumor found in the optic nerve. It was removed intraorbitally without removing the eyeball, but later the eyeball became so irritable that it became necessary to remove it. Since that time there has been no irritation whatever, and that was about a year ago. There are no evidences anywhere else in the system of Recklinghausen's disease. The left eye is normal in every particular, and microscopic diagnosis as determined by the Army Medical Museum was a definite glioma.

This case is reported merely to add one more to those without complications which have already been reported.

DR. ALAN C. WOODS, Baltimore: My reason for discussing this paper is to protest against the opinion voiced by Dr. Bailey that these tumors should preferably be operated on by the ophthalmic route. Patients with orbital tumors are seen in the Johns Hopkins Hospital in both the ophthalmologic and the neurosurgical service. Those operated on in the ophthalmologic service are operated on either by the frontal ophthalmic approach or by the Krönlein route. Many tumors operated on by these procedures in the past were found to have intracranial extensions which could not be demonstrated before operation, and complete removal of the tumor was therefore impossible by either approach. After several such disastrous experiences, I now make it a practice to refer all patients with tumors deep in the orbit to the neurosurgical service; they are then operated on by the transfrontal route, which readily permits complete removal of deep orbital tumors and also affords a surgical approach to the intracranial extension if one is present. I believe the ophthalmic approach should be limited to tumors in the anterior portions of the orbit with which there appears to be no possibility of intracranial extension.

DR. FREDERICK A. DAVIS, Madison, Wis.: With regard to the lesion of the disk mentioned by Dr. Levitt, I have referred in my paper to 2 cases. The growth in 1 case was a true glioma of the retina. It was reported by McLean from the Johns Hopkins Hospital, and so far as I know it is the only true glioma of the retina on record. Skala also reported a similar case, although I believe he diagnosed the growth as a neurofibroma.

With regard to Dr. Woods's remarks, it is interesting that the brain surgeon should endorse my method of approach, which is contrary to the approach of such a surgeon.

The transfrontal procedure was used in 2 of the cases reported here—cases in which the chiasm was involved. Naturally if the chiasm is involved, it is useless to attempt to remove any growth in the orbit, because the condition is hopelessly inoperable. The best that one can do is to decompress the optic foramen by removal of its roof and make an incision in the sheath of the nerve. This was done in 1 case. This procedure has been reported as having been used elsewhere, with the result that 1 patient has retained some vision for a period of ten years. I realize that the glioma grows very slowly. It may take ten to twenty years for it to develop into a full-sized tumor, so that during that period if the optic nerve enlarges slight vision may be preserved for a considerable period.

I brought out one point in the paper that time did not permit reading; otherwise Dr. Woods might not take exception to my approval of the anterior approach. I believe that these tumors of the optic nerve are merely a manifestation of a generalized systemic disease. Therefore, other lesions presumably exist in the chiasm, in the temporal lobe of the brain (as was seen in 1 case reported here) and perhaps in the optic nerve; so that after all, the operation on the orbit is nothing more than

a palliative measure to relieve exophthalmos and preserve the globe. I do not believe that the glioma of the optic nerve extends back into the cranial cavity necessarily. Such extension, of course, is possible, but in following the history and autopsy reports of most of these cases it seems that other lesions already exist in the brain, just as in neurofibromatosis or peripheral Recklinghausen's disease there may be retinal foci in the central nervous system.

MALIGNANT MELANOMA OF UVEA: SO-CALLED SARCOMA; II. PROBLEMS IN DIAGNOSIS. DR. T. L. TERRY, Boston.

This article is published in full in this issue of the ARCHIVES, page 989.

EXPERIMENTAL STUDIES OF OCULAR TUBERCULOSIS: STUDIES IN THE IMMUNE-ALLERGIC RABBIT. DR. ALAN C. WOODS and DR. EARL L. BURKY, Baltimore.

The material for this study together with some additional material will be published in later issues of the ARCHIVES as three separate articles, nos. IV, V and VI of the series entitled "Experimental Studies of Ocular Tuberculosis."

DISCUSSION

DR. M. HAYWARD POST, St. Louis: As a clinician, primarily, this painstaking and elaborate piece of investigation which is now under development by the authors of this paper and their colleagues appeals to me, especially from the point of view of its practical application in the diagnosis and treatment of suspected ocular tuberculosis.

On Oct. 19, 1937, Dr. Woods read a paper before the Interstate Postgraduate Medical Association of North America on the subject, "The Problem of Ocular Tuberculosis." His thesis was developed under two principal subdivisions: (1) the diagnostic problem and (2) the therapeutic problem. He pointed out therein that the diagnosis depended on: (1) the character and course of the ocular lesion, and the correlation of this with similar lesions, previously shown to be tuberculous by histologic examination; (2) an exclusion of other possible etiologic factors; (3) a study of the general tuberculous status of the patient, and (4) the reaction of the patient to tuberculin.

The immediate problem now under consideration, as related to diagnosis of the ocular lesion, is concerned chiefly with the fourth of these considerations, that is, the proper evaluation of the reaction of the patient to the intradermal use of tuberculin.

Likewise, turning to the therapeutic problem, my interest in this connection is centered on the value of the Mantoux test as indicative of the progress and prognosis of the tuberculous lesion in the affected eye.

It is well recognized that the diagnosis of ocular tuberculosis leaves much to be desired. This is easily appreciated as one studies patients suspected of having ocular tuberculosis and also as one peruses numerous clinical reports and finds that the diagnosis of ocular tuberculosis varies

from as low as 5 per cent of all ocular lesions in some clinics up to as high as 50 per cent in others. The European clinics, where less attention is paid to focal infection than in America, are responsible for the higher figures.

While there are many tests in use for determining the presence of tuberculosis, the Mantoux test, or the intradermal injection of old tuberculin or, more recently, purified protein derivative, is the test which is probably the one that is most frequently relied on. The reaction, although it rests on well established grounds with regard to its reliability as an indicator of dermal sensitivity to tuberculoprotein, apparently leaves much to be desired relative to its value as an index of tuberculosis of the eye. Indeed, numerous clinical investigations have previously indicated the fallibility of this test in this regard. As pointed out by the authors, clinical investigations along this line at the Wilmer Ophthalmological Institute have been most disappointing. Also, in clinics scattered throughout both Europe and America many patients with histologically proved ocular tuberculosis have failed to show positive reactions to the Mantoux test. Such considerations have led Dr. Woods and Dr. Burky and their colleagues to undertake the experiments of which the most recent developments have just been reported.

It would seem that the status as to previous tuberculous infection, with its consequent allergy and immunity, met with in the great majority of instances of ocular tuberculosis encountered in the human being has been simulated closely in the immune-allergic rabbits used in these experiments. It appears justifiable to assume that the reactions observed in these animals are, therefore, applicable to the average clinical patient. Such being the case, in the light of the observations reported in this paper, the conclusion is forced on one that the intradermal test in cases of suspected ocular tuberculosis indicates only the cutaneous sensitivity and in no degree the ocular sensitivity and consequently is of little diagnostic value as to the nature or activity of the ocular lesion, except in so far as it may be useful for the determination of a previously existent state of allergy and immunity in the patient.

With regard to the second phase of this subject, namely, the therapeutic problem, the situation seems to be but little better. When one asks whether the Mantoux test repeated during the course of tuberculin therapy for ocular tuberculosis gives any indication of the degree of ocular activity, one is forced to the conclusion that at best it can give only a slight clue as to the ocular sensitivity, and since ocular sensitivity does not conicide at all times with ocular activity, it can be of little, if any, practical value.

In the light of such conclusions, one may well ask oneself of what possible value, then, is the use of tuberculin in ocular tuberculosis. One finds the answer in that these experiments do show the desirability of reducing the ocular sensitivity to a minimum in order to prevent, to as great an extent as possible, the violence of the initial response and subsequent course of the ocular infection. This is true even though the ultimate outcome is uninfluenced thereby, provided the initial attack is not of such virulence as to overcome that immunity which is present, permitting free growth of the bacteria and thus creating a high degree of ocular sensitivity and tissue destruction.

That the use of tuberculin, even in small doses, may accomplish such a desensitization at the site of the lesion is strongly indicated both by clinical observation and by certain experimental studies of analogous conditions, as indicated by Dr. Woods in the paper previously referred to. He pointed out therein that the well known Schwartzman phenomenon shows that "a prepared inflammatory focus caused by a nonspecific inoculum, such as a colon bacillus filtrate, will be activated by intracutaneous injection of tuberculin—an example of the predilection of a highly diffusible tuberculin to filter out and be deposited in a local inflammatory process," a point now under investigation by Rich and his co-workers.

Finally, an exceedingly important observation brought out in this work should once more be emphasized, namely, that the ultimate outcome is dependent on the state of immunity rather than on the state of sensitivity and that immunity appears to be definitely increased by a preceding attack of tuberculosis, once the quiescent state has been reached.

Dr. Albert L. Brown, Cincinnati: Dr. Woods and Dr. Burky have studied the ocular reactions of infected animals and by successively varying the combinations of bodily and local injections have clarified several ideas of tissue reactions in the rabbit eye. Immune-allergic animals with varying degree of general and of ocular immunity and sensitivity have been given intraocular injections of large doses of live tubercle bacilli.

Perhaps too much credence has been placed on reactions following intraocular injections of purified protein derivative in the inflamed and quiet fellow eye as an indication of ocular sensitivity. My personal experience has shown that the injection of any material, even a little air or salt solution, will usually increase the reaction in a highly inflamed eye, whereas it produces only slight, transient irritation in a normal globe. This is offered merely as a suggestion, and since I have not seen the essayists' animals, I cannot judge them fairly.

Since the inflammatory reactions were obtained by intraocular injections of live organisms and in many instances massive doses, any suggestion of analogy to the clinical disease must be examined carefully. How far can the results of these experiments be applied to clinical cases? It is well known that ocular tuberculosis occurs most frequently in patients with no manifest bodily infection. Surveys of inmates of sanatoriums for tuberculous persons show an extremely low percentage of ocular involvement. Furthermore, as has been stated, dependence on intravascular injection of organisms for ocular infection in animals would create a shortage of cage space. Since human ocular tuberculosis is assumed to arise from a systemic infection, it is difficult to conceive of sufficient organisms finding lodgment in an eye to cause a profound inflammation, especially in a presumably immune-allergic person, when it is necessary to inject such a massive dose directly into the eye of a similarly prepared animal. Why should a focus, innocuous for all other organs, supply organisms of sufficient quantity to infect an eye and occasionally both eyes without any other apparent sign of bodily involvement?

Another mechanism that might be considered is the response of a sensitized eye to the circulating products of the tubercle bacillus. This point of view eliminates the fortuitous lodgment of organisms in the

eye and explains the failure of most observers to find organisms in ocular tissues in proved cases. The tubercle bacillus has properties that make this type of investigation difficult. Tuberculin and even the whole dead organism are weak antigens, and it requires the live organism to produce any demonstrable reactions. The essayists report the most systematic attempt to qualify tissue reactions as one must know them to evaluate treatment.

DR. ALAN C. WOODS, Baltimore: In regard to Dr. Post's theory on the use of tuberculin, I still feel that tuberculin has a definite therapeutic place as a desensitizing agent. My co-workers and I have a number of experiments, as yet unfinished, which will apparently bear out the value of keeping the ocular sensitivity at a low level both in controlling the disease and in preventing its recurrence.

In regard to Dr. Brown's first question about drawing clinical interpretations from these experiments, it is the last thing I wish to do. I believe that the ocular disease produced in the rabbits in this study is the closest analogy to human tuberculosis that can consistently be obtained. I admit the mechanism of production is entirely different. But if one employs intracarotid injections of the organisms and uses large enough doses, most of the animals die of cerebral emboli. If small doses are given, ocular tuberculosis results in 1 out of 100 rabbits.

One other point Dr. Brown brings up that is pertinent is the question of how enough organisms are going to get into the eye to cause intraocular tuberculosis. We use a virulent human strain which causes some limited disease in the rabbit and does not kill it. It lives long enough for us to get prolonged observations, and we use the minimum dose that will surely produce ocular tuberculosis.

The marked differences between that and human tuberculosis were emphasized by Dr. Post. They bear out our clinical observation that organisms can be recovered from rabbits with known ocular tuberculosis. When there is known tuberculosis, tuberculin (purified protein derivative) gives the weakest sort of reactions in many animals or sometimes even almost negative reactions. Did we not know the nature of the ocular disease of these animals, the cutaneous Mantoux test would give little or no information about its actual nature.

A STUDY OF EXFOLIATION OF THE LENS CAPSULE (GLAUCOMA CAPSULARIS): FORTY CASES OF EXFOLIATION. DR. RODMAN IRVINE, Los Angeles.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

CATARACT FOLLOWING THE INHALATION OF PARADICHLOROBENZENE VAPOR. DR. MILTON L. BERLINER, New York.

This article is published in full, with discussion, in this issue of the ARCHIVES, page 1023.

OBSERVATIONS ON THE EYE AND THEIR SIGNIFICANCE IN TRAFFIC COURT CASES. DR. LOWELL S. SELLING, Detroit.

Examinations are given in the traffic division of the psychopathic clinic, recorder's court, to traffic offenders and accident-prone drivers,

consisting of mental, physical and psychologic tests. These same tests were given to a control group. The ophthalmologic examination includes tests of color vision, depth of vision, sensitivity to glare, visual acuity, visual fatigue, depth perception and judgment of speed and distance. Ophthalmoscopic examinations are performed also. Eight hundred persons, of whom about 100 were controls, were studied in this manner, particular attention being paid to the horizontal axis of the visual fields, results of glare testing, color blindness and ophthalmoscopic examinations. These factors are of significance with regard to the operation of motor vehicles.

DISCUSSION

DR. ARTHUR J. BEDELL, Albany, N. Y.: Dr. Selling sent his paper to me, and after studying it I am convinced that the special points to which he has drawn attention, organic disorders of retinal function, color vision, "visual span" and glare sensitivity, are important phases of the ocular examination of automobile drivers.

It was reassuring to have Dr. Selling confirm my previously stated impression that vitamin A deficiency seemingly has no demonstrable effect on vision in the dark. Although his clinical apparatus does not simulate actual working or driving conditions, his report is worthy of serious consideration, but before enduring conclusions can be reached, it seems advisable to have as controls a much larger number of automobile drivers from several different localities.

The author's observations on color defects are interesting and well substantiated by ophthalmologic experience. The intensity of light is frequently the guide by which a color-blind automobile driver distinguishes between the stop and go signals.

Just before I left for several meetings in London, I made a personal contact with the authorities in charge of the New York State Bureau of Motor Vehicles, to determine, if possible, some of the ocular reasons for automobile accidents. Through the kindness of Commissioner Carroll E. Mealey and the active cooperation of Mr. Robert J. Maloy, I was permitted to check the reports which are now being tabulated in the department at Albany. Eventually, the segregated material will be of great assistance to those engaged in investigating and analyzing accidents as well as to those working to prevent them.

Perhaps the most important observation made from the available records is that drivers with markedly defective vision in one eye had an accident on the side with the better vision in 50 per cent of the cases and that the other 50 per cent were on the side of the poorer eye. But here again the cross section of operators and accidents is too small to even predicate a suggestion of a statistical formula. The findings have a distinct bearing on the author's perimetric studies and conclusions and may in fact lead to the intimation that the mental condition of the driver is, after all, one of the essential factors in safety.

The work this section has done in outlining the visual requirements has met with widespread approval, and the standards have been adopted by several states. It is therefore appropriate to appraise Dr. Selling's contribution at this time, for both the walkers and the riders of the world are anxious to be protected from sudden death and injury.

DR. ALBERT C. SNELL, Rochester, N. Y.: In the Detroit traffic court clinic, Dr. Selling has an unusual opportunity to make some valuable contributions to a better understanding of traffic problems, particularly those having visual and psychologic aspects.

Many problems are suggested in this paper, but Dr. Selling has limited his discussion to retinal functions, especially to sensitivity to glare. In his conclusions in regard to this he stated that the "solution is still up in the air." This statement seems to be true for several reasons: 1. The term glare has not been precisely defined. 2. There is uncertainty as to the exact elements of the function of vision which are being tested by the glarometer. 3. There is no standard for glare sensitivity by which to determine the normal or efficient operator and the inefficient or dangerous operator.

The term glare may mean many different things to as many different persons. According to the Century Dictionary, "glare" is "a bright light or a confusing and bewildering light." Another definition, taken from Collier's National Encyclopedia, states that "glare is a blinding sensation caused by excess brightness of light in the field of vision, by extreme contrast between a light source and its surroundings or by continued exposure of the eye to a light source." Some of the lighting engineers have defined glare as "light out of place." None of these definitions present a satisfactory scientific or working basis. Glare is certainly something more than a bright light or excessive brightness, since in daylight, when the intensity is 1,000 candlepower or more, glare is a negligible factor if it includes the idea of a blinding sensation. However, brightness is a necessary sensation for seeing. Glare might be defined as a disturbance of visual sensation caused by excessive aberrant rays of light.

In the use of the so-called glarometer, I am at a loss to know exactly what physiologic function of vision is being measured or tested. It would seem that to place the eye in or before a dark box and then suddenly to expose it to a high intensity of light would produce a sort of shock to the receptor elements of the retina. Then as this sudden shock to the dark-adapted eye is followed by gradual decreases in intensity of illumination, we probably have a test of the power of adaptation. But since the test object is placed at the center of fixation and also of illumination, this test seems to be one of speed of recovery from retinal shock, fatigue or exhaustion for variable decreasing intensities of light. The test is probably not that of adaptation of the macula, as this sense is absent here according to the belief of physiologists.

The physiologists make a distinction between adaptation and fatigue. Adaptation, as defined by Adler, is "a decline in the excitability caused by the stimulus"; "fatigue is a decline in the activity of an organ caused by its previous activity." The tests as made by the glarometer seem to combine both of these factors, adaptation and fatigue. I wish that Dr. Selling could make clear the relation between adaptation, fatigue and glare.

Evidently there is some relation between the adapted eye, contrast, the intensity of illumination, the length of exposure for various intensities and the time period necessary for recovery of a normal visual function. It would be helpful if some of the scientific workers could establish some definite formula by which all these factors when taken

into consideration in their relation to glare would give a quantitative measurement by means of which glare sensitivity might be made practical for the determination of a person's capacity or efficiency to operate a motor vehicle.

Dr. Selling speaks of an "adequate function," but this is not defined, and from the data which he presents I find no clue or answer to the question, "What is an adequate retinal function?"

The test object looked at in Dr. Selling's tests require an approximate visual acuity of 20/60. It seems to me that this low acuity destroys to some extent the value of the test, since visual acuity is acknowledged as an important factor in a person's tolerance to glare. Lauer states as a fact that "the poorer the visual acuity the lower the tolerance to glare." It seems to me that glare tests should be made with a test character which corresponds to a person's best acuity while wearing the proper correcting lenses.

The complexity of the problem is obvious and still unsolved. For this reason the subject of glare was omitted from the report of the Committee on Visual Standards for Operators of Motor Vehicles, which was submitted to this section last year. This committee found no standard for normal and no satisfactory instrument by which to measure glare sensitivity so that a person might be classed as a hazard in operating a motor vehicle.

DR. LOWELL S. SELLING, Detroit: I tried to limit my paper to a small area in this matter of testing the eyes of drivers, and both discussants have opened up rather vast fields which I feel I should not take into consideration at the present time.

There are only one or two points I want to touch on. First of all, in answer to Dr. Bedell's comments I should like to say that the reason a special perimenter is used is because one can eliminate cheating. With the large band perimeter one can detect certain types of cheating, the flick of the eye which the observer occasionally misses.

Another thing that I am trying to eliminate is the extra factor of visual acuity from these tests, because I have a grave question in my mind as to the standards of visual acuity for drivers. I hope that sometime I can give the material to the opthalmologists when I have it in shape. It is rather extensive. The factor of visual acuity is eliminated by having a device which is a meter away from the eye. In other words, a rather gross piece of apparatus is used instead of something to detect fine stereoscopic changes.

Dr. Snell opens up a wide field. He is right in saying that glare is not defined. With the apparatus used for these tests I get a correlation of about 0.62 of night accidents due to glare sensitivity. I did not give that figure in my paper because I do not think it is substantiated by a sufficient number of cases. Nevertheless, this apparatus does similate what goes on on the highway. In other words, when a car comes at one with very bright light, a person is reacting to that bright light, and the accident occurs while the retina is recovering. I am trying to find some sort of measure of recovery of the retina which can be reported; in other words, a symbol instead of a word. It is not a matter of visual acuity; if one has a large object the light will show through it even at low intensities and be easy for the person to see. Perhaps ophthalmologists have never given thought to the

question of whether visual fields are not limited by the type of hats that women wear. I think that in many cases women get into a good deal of trouble while driving because one eye is covered. Another matter of glare is probably mechanical. Many persons sitting in the back seats of automobiles throw the headlights directly into the eye of the oncoming driver, so that one car with similar illumination to another causes actual glare when the lights are uptilted.

THE PLACE OF OPHTHALMOLOGY IN THE PREVENTION OF BLINDNESS. DR. F. PARK LEWIS, Buffalo.

The purpose of the ophthalmologist is to preserve sight by preventing conditions which injure the eyes and by repairing damage when it has occurred. Prevention is the watchword of all scientific progress today. Only organized movements can be largely successful, and these have been mostly of a popular nature. In what way are medical societies actively associated with preventive measures? What is the present status of ophthalmia neonatorum throughout the country? Are facilities provided everywhere for the prompt hospitalization of the infected infant? The remaining 8 per cent of the ocular mortality due to ophthalmia neonatorum is too high. What is being done to prevent keratitis interstitialis? Are clinics adequate and effective? What facilities exist in sparsely settled districts to secure early diagnosis and good ocular surgical treatment? How can they be improved? What can be done about glaucoma? These questions are discussed and measures advised.

DISCUSSION

DR. EDWARD V. L. BROWN, Chicago: Organized work for the prevention of blindness has been carried on in this country for some thirty years, and Dr. Parks Lewis has been right in the forefront all of the time. The first efforts, around 1910, were directed against gonorrheal ophthalmia neonatorum and were rewarded with a wonderful measure of success.

Sight-saving classes came next (1915 to 1920). In these, a child who has lost about a third of his vision and cannot read ordinary school books regularly makes full progress in school. He is given books with 24 point print. Letters such as T, L, O, etc., measure 3/16 (0.48 cm.) of an inch high. There are now hundreds of such classes in the United States, all within the framework of the regular public school system.

Next came campaigns against industrial and Fourth of July injuries. They bore great fruit. From 1920 to 1925 came a special attack on trachoma, lately vivified by the possibility that sulfanilamide may cure the disease. After this came social service help in glaucoma clinics (1925 to 1930). Lastly, the ravages of syphilis of the eye have received special attention along with the country-wide campaign against syphilis.

Thus, in all there have been about five major moves in connection with ophthalmia and sight-saving classes, trachoma, accidents and syphilis. Great good has been done. But there is one unsatisfactory aspect to this work. Laymen have raised the funds and directed policy through lay executives, administrators, social workers and publicity experts. Medicine has not always insisted on, nor has it been given, its

proper place. It has furnished technologic help. The issue is probably basic. Medicine is individualistic. It stresses the relation of the patient to the physician. Prevention work tends to be collectivistic. A child can read only so much on the test chart, so must go into a sight-saving class. It is better to have three in the class who do not belong there than to fail to have one in the class who should be there. Some lay workers and some physicians feel that all persons with myopia of over 4 diopters should be put in sight-saving classes despite their normal vision.

A medical executive once overstressed the importance of diet in trachoma. On the basis of records admitted to be very defective, syphilis has been publicized as a cause of ocular disease in a percentage which many think is a gross exaggeration.

Much of the fault lies at the ophthalmologist's door. It has not been determined accurately how much ocular trouble is due to syphilis. The ophthalmologist has not been active in accident prevention work. He tends to protect colleagues who are remiss in the use of silver nitrate in the newborn. He does not realize as fully as he should all that is involved in simple glaucoma, and he should more sympathetically explain everything to the patient. The ophthalmologist himself is the best social service worker.

Dr. Conrad Berens, New York: So many years of Dr. Lewis' long and useful life have been given to the fight for the prevention of blindness that there is no one in this country who can speak more authoritatively than he. Dr. Lewis has briefly sketched the origin of the movement for the prevention of blindness in this country, but with characteristic modesty he has not mentioned his great part in this. If it had not been for his pioneer organization work in conjunction with Miss Louisa Lee Schuyler, the National Society for the Prevention of Blindness might still be unheard of and the International Association for the Prevention of Blindness might never have been born.

Likewise, Dr. Lewis played an important role in the early work of this section in the movement for the prevention of blindness, for he was chairman of the first committee to deal with preventive ophthalmology. It was largely through his influence that some of the states organized committees to cooperate with the National Society for the Prevention of Blindness.

Dr. Lewis has raised many points which require the active cooperation of all ophthalmologists in the United States and other countries. Therefore, a word asking for greater cooperation between practicing ophthalmologists and agencies, both state and national, devoting their activities to the prevention of blindness, seems justified.

I am delighted that Dr. Lewis introduced the question of optometry, especially since the opticians, optometrists and ophthalmologists cooperated so successfully in New York this spring in sponsoring joint legislation which will do much to prevent racketeering practices that are detrimental to the public health. I was informed by an authoritative source that 66 per cent of the glasses prescribed in this country are prescribed by optometrists. There is no doubt that they often see patients with serious ocular diseases which may result in blindness earlier than do ophthalmologists. The medical profession should certainly strive to work out a solution concerning the ethical relations

between themselves and optometrists so that the public health will not be jeopardized. I urge every member of the American Medical Association to cooperate with the American Committee on Optics and Visual Physiology, which is endeavoring to find some solution to this important unsolved problem. I only wish that Dr. Lewis had had more time so that he could have discussed this question in greater detail, the solution of which would help to prevent much unnecessary blindness.

There is no doubt that ophthalmologists and medical men in general have been remiss in their duty in educating the public concerning important medical matters. Every ophthalmologist here can help in the movement for the prevention of blindness by heeding Dr. Lewis' suggestion concerning the education of the public. While public lectures, as suggested by Dr. Lewis, are valuable, they reach such a small group; the radio should be used as a means of carrying into every home in the United States authoritative medical knowledge concerning the prevention and treatment of ocular diseases. I am glad to say that the Section on Ophthalmology of the New York Academy of Medicine has appointed a publicity committee to arrange for radio addresses to be given over a national network. During the past few years, addresses on the public health aspects of ocular diseases have been given from time to time which were sponsored by the New York Academy of Medicine at large but not by the Section on Ophthalmology. I believe that this condition holds true for the American Medical Association, and I wonder whether it would be desirable for this section to appoint a committee on ophthalmologic public relations to deal with needed publicity and other important matters affecting the ophthalmologist's relations with the public.

Physicians in general, and possibly especially ophthalmologists, often fail to recognize the great opportunities to educate the public in matters pertaining to the prevention of blindness through cooperation with organized medicine. They should take an active interest in their county medical societies and in committees of other organizations which actively participate in public health matters. It is only by broadening the contacts of ophthalmologists with those interested in many fields of endeavor that the important message to be given by opthalmologists concerning the prevention of blindness can be utilized as widely as it should be. For example, if the knowledge of the most competent ophthalmologists is not available to the department of health in our cities and to the leaders of industrial organizations in the country, methods of preventing blindness from certain diseases might never be initiated. Delay might also occur in the application of the most important new developments in the prevention of certain ocular infections. Moreover, if ophthalmologists do not make contacts with industry, the appalling loss of time and sight through industrial accidents may not be so rapidly lessened. As Dr. Lewis has pointed out, machinery for preventing industrial accidents is at hand, but in some plants the knowledge is not used. This again is frequently caused by lack of information or proper stimulation.

Again if the ophthalmologist does not present papers before societies other than those dealing with his own special branch, he may not be able to carry a message to his fellow practitioners which might lead to the early diagnosis of certain conditions, for example, amblyopia in children who have little evident deviation of the visual axes.

There is no doubt that glaucoma is still an important unsolved problem, and I am glad that Dr. Lewis has again stressed the importance of early treatment of this disease and the discovery of the cause so that prevention may be more scientific. I heartily agree with him in his suggestion concerning research and hope that when the time comes to make these correlated studies in association with proper groups, the study of chronic infection in relation to the problem will not be neglected.

The message brought by Dr. Lewis has been so stimulating that I am sure each one present will feel compelled to carry out his suggestions. May the promises made today to cooperate in preventing blindness not be broken through the pressure of routine duties, because by such efforts much can be done to prevent that part of blindness that Dr. Lewis says "need never have happened"!

Dr. F. Park Lewis, Buffalo: My purpose in bringing this matter before this section was not to tell things that all know but to see whether some method might not be developed by which as a body of ophthalmologists these unnecessary losses might be minimized. It can de done only by an authorized method.

The earlier part of the century, I think, there were committees of members of the American Medical Association formed in every state, the purpose of which was to work through the county societies down to the ultimate medical unit. The result did much in controlling ophthalmia neonatorum. The same thing might do much toward preventing these other conditions to which I have referred.

There was not time to enter into any method of detail as to the manner in which this might be done. I simply want to ask the section to take it under advisement, because the members know better than I do how it might be carried into effect.

Progressive Myopia, Keratoconus and Keratoglobus: Their Causation and Treatment. Dr. Julian Y. Malone, Milwaukee.

Progressive myopia, keratoconus and keratoglobus are grouped together, because each condition represents a stretching of some portion of the skeleton of the eyeball. In 22 per cent of 574 cases studied, myopia was of the progressive type. This percentage was reduced to 1.7 by treatment, showing effective control. There is evidence in favor of an endocrine imbalance as the main etiologic factor in progressive myopia, keratoconus and keratoglobus.

DISCUSSION

Dr. William L. Benedict, Rochester, Minn.: In every case myopia is, naturally, progressive at some time. However, in the majority of cases the myopia is on a developmental basis, and there is little, if any, degenerative change; the eye might be considered as a normal one well adapted for close work. The hereditary background of the patient apparently plays the dominant role in producing the condition. The group of myopic eyes to be considered as diseased eyes and therefore possibly to be affected by treatment is relatively small.

Dr. Malone's results with epinephrine are much better than those reported by Dr. Weiner in 1931. The myopia progressed in approximately 20 per cent of his cases in spite of the use of epinephrine and

full correction. This figure is about the same as that given by Dr. Jackson for his cases in which the treatment consisted only of full correction. My percentage is about the same.

I have confined the use of epinephrine to the so-called malignant type of myopia and to patients with keratoconus. My results have not been as good as Dr. Malone's. In many cases the myopia has continued to progress in spite of the use of epinephrine and full correction. However, I feel that there is, if anything, less degenerative change in the eyes in which epinephrine has been used than there might be had it not been employed. However, this is pure speculation on my part.

I see a relatively large number of patients with keratoconus every year, many of whom come because of progression of the condition. A number of these have been using epinephrine for some time without noticeable effect. With existing knowledge of the physiology of the cornea, one would consider this the ideal type of case for local treatment to be effective.

While one might agree that it is through the endocrine system that myopia is initiated, I do not believe lack of epinephrine can be blamed. I see on the one hand patients with tumor of the adrenal gland and a tremendous amount of epinephrine circulating in the blood and on the other hand patients with Addison's disease and practically inactive adrenals. Neither group of patients is distinguished by any refractive change. I feel that if epinephrine has any effect it is through local action and not in supplanting a deficient hormone.

I believe that all agree that in both keratoconus and myopia the greatest factor is heredity. It is discouraging for one interested in therapeutics rather than in heredity to have under his care a mother and two daughters all with keratoconus, as has been my experience during the last year.

I believe in full correction and sensible restriction of close work in cases of myopia of the more severe type. Great mental harm can be done a child or a young adult by overemphasis of myopia as a physical defect or by advising a tutor or special classes for them or by telling them not to go to college or not to study medicine.

Dr. Meyer Wiener, St. Louis: Progressive myopia does not necessarily mean a stretching of the scleral or corneal walls but may consist of a natural proliferation of cell tissue of the walls due to overstimulation. This would eliminate, then, change in the intraocular pressure as a factor in the production of certain types of myopia. I have in mind the most common type of myopia, or that due to heredity.

Just as one is predisposed to inherit features and structural tendencies from one's progenitors, so is one likely to inherit a similarly shaped eye.

I agree with the author that the age incidence is most important as a time factor but not as a cause. Sexual development may play some part in influencing the growth of the eyeball, although this is not borne out by following cases of known retardation of growth of the genital organ.

It is true that general endocrine studies, including sugar tolerance tests, have not thrown any particular light on the subject. I believe, however, that basal metabolism tests have a definite value. A large

percentage of persons with progressive myopia have a lowered basal metabolic rate. When this is brought up to a proper level it seems to have a definitely beneficial effect in checking the progress of the myopia.

I have not been as fortunate as Dr. Malone with either the small number of doses used or the length of time the drug is used. A minimum of three or four instillations daily are recommended and kept up for at least a year. The patients are then allowed to reduce the number of instillations gradually, all the while being kept under observation at intervals of three to six months.

I have never seen true axial myopia improve, although in the majority of cases the myopia is either checked or slowed up.

In every case of conical cornea caught in the early stage, treatment has not only arrested the progress of the condition but has improved the refraction and vision.

Strenuous exercise is always advisable. It makes little difference, in my experience, how much or how little the eyes are used or whether or not the glasses are worn constantly, so long as the use of the eyes does not interfere with the patient getting plenty of exercise, preferably out of doors.

Naturally, a well balanced diet is essential. The work of Blackberg and Knapp is not convincing as far as it relates to true axial myopia and not to corneal involvement of corneal disease. It seems to me that in those cases of conical cornea in which improvement is reported the cornea is weakened either because of low vitamin D intake or possibly as the result of lenticular changes. I cannot visualize a rigid wall such as the sclera as actually shrinking.

The use of epinephrine for influencing the course of progressive myopia was actuated by the report of Paul Pontius, who first observed the complete reduction of the bulge in ectasia of the cornea by instillation of a 1:1,000 solution of epinephrine hydrochloride. This phenomenon has been confirmed by me both experimentally and clinically over a period of many years. It has been proved by many scientific observers that epinephrine, even in minute doses, increases the metabolism of practically all tissues.

If epinephrine increases the metabolism of corneal tissue and exerts a beneficial effect on it, why can it not have the same influence on the scleral tissue, which is similar in its structure?

DR. T. L. TERRY, Boston: I have seen a relatively large number of patients with conical cornea, and of that group there are comparatively few who are in the age period at which one would expect advancement of conical cornea to occur. During the last year a marked and sudden increase in the amount of conical cornea present developed in 2 patients.

In one instance the patient had a severe fit of sneezing, lasting half an hour. This occurred at night. The following day he had considerable disturbance of vision in each eye associated with a bilateral acute exacerbation, one might say, of the amount of conical cornea. The patient at the time was taking large doses of vitamin A, vitamin D and calcium. In the other instance the increase in the cone was limited to one eye. Although the patient had been on a high vitamin and calcium diet, a local physician advised a diet poor in calcium. Four days after the institution of this diet the conical cornea suddenly developed.

Whether or not these acute exacerbations have any real significance, I cannot yet determine. Whether coughing or sneezing could have some effect on a weakened cornea, I do not know.

Careful studies to discover the evidence of abnormal metabolism, abnormal endocrine disturbances and hereditary ocular bases for the disease in a number of young persons who may be having an increase in the amount of conical cornea has revealed nothing of significance.

The relation of this condition to myopia does not seem very obvious. This acute exacerbation does not appear consistent with instances of progressive myopia.

INTERSTITIAL KERATITIS CAUSED BY SPECIFIC SENSITIVITY TO INGESTED FOODS. DR. ABBOTT M. DEAN, DR. F. W. DEAN and DR. GUY R. MCCUTCHAN, Council Bluffs, Iowa.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

Obituaries

HOWARD McILVAIN MORTON, M.D.
1868-1939

Dr. Howard McIlvain Morton died on July 19, 1939, at Vincentown, N. J., at the age of 71. He was born at Chester, Pa., on May 23, 1868, of Swedish ancestry. He attended high school at the Maplewood Institute at Chester, later entering Lafayette College, Pennsylvania, where he received the degree of Bachelor of Science in 1888 and of Master of Science in 1889. He was graduated from the University of Pennsylvania Medical School in 1891 and was an intern at St. Luke's Hospital, Bethlehem, Pa., in 1890 and 1891. He engaged in general practice for only a short time before proceeding to the Royal London Ophthalmic Hospital (Moorfields) and to the Charité clinic in Berlin for training in ophthalmology. He located in Minneapolis in 1891, where he immediately began the practice of ophthalmology and otolaryngology. Dr. Morton was active in the social and musical circles of Minneapolis. He became prominent in medical societies in Minnesota and was one of the founders of the Minnesota Academy of Ophthalmology and Otolaryngology, being its first president He made frequent contributions to the medical literature and had a special interest in the development of perimetry. He wrote extensively on this subject, being the author of the chapter on perimetry in the American Encyclopedia of Ophthalmology. He also devised the Morton perimeter. For a time he was professor of ophthalmology at Hamline University, the medical department of which was merged with the University of Minnesota.

During the World War, Dr. Morton enlisted for active service and was stationed the greater part of his time at Fort Oglethorpe, Ga. He held the rank of major. At the end of the war he resumed his practice in Minneapolis and was exceedingly active until his health began to fail. In 1931, with Mrs. Morton, he returned to Vincentown, N. J., to make his home with Dr. Rush K. Morton, his brother, and his sister, Mary W. Morton. He was a member of the Minneapolis Club, the "Skylight Club," Delta Tau Delta and Phi Alpha Sigma. He was an active Episcopalian. A gifted speaker, he took great interest in municipal affairs and was a generous literary contributor to the many organizations of which he was a member.

In 1891 Dr. Morton married Lucretia Gail Jarvis, who survives him. There were no children. Dr. Morton had a host of friends in his profession and in the community of Minneapolis and in Minnesota. He was an ardent amateur ornithologist and took great interest in developing a lovely summer home, where he indulged in landscape gardening and frequently entertained his many friends and colleagues. He was a most conscientious and careful practitioner in his specialty;

HOWARD McILVAIN MORTON, M.D.
1868-1939

loyal to the interests of his profession and its advancement and always courteous and ethical in his professional relations, he commanded the universal respect of his associates. Those who knew this chivalric savant will always remember him for his ardent enthusiasms, his inexhaustible amiability and his courteous old-school gentlemanly qualities.

FRANK E. BURCH.

Book Reviews

Anales argentinos de oftalmologia. No 1, vol. 1, 1939. Rosario, Argentina. Hospital Centenario.

The ophthalmologists of Rosario, Argentina, have founded a Society of Ophthalmology in the Littoral. The transactions of this society will appear in the *Anales argentinos de oftalmologia,* which will also publish the scientific papers from the department of ophthalmology of the University of Rosario. This new magazine will be issued quarterly. The editor is Prof. Carlos Weskamp. The first copy contains a number of original articles and a review of the European journals.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 4-6, 1940.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: New York. Time: June 10-14, 1940.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Nadeau, 122 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: The Gateway Inn, Land O'Lakes. Time: June 1940.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. A. Shultz, 303 N. Main St., Rockford, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
Secretary: Dr. M. P. Spearman, 1001 First National Bank Bldg., El Paso, Texas.
Place: El Paso, Texas. Time: Nov. 9-11, 1939.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Virgil Payne, Pine Bluff.
Secretary-Treasurer: Dr. Raymond C. Cook, 1005 Donaghey Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Melville Black, 424 Metropolitan Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Shirley H. Baron, 309 State St., New London.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Grady E. Clay, 384 Peachtree St. N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. D. C. Montgomery, 301 Washington Ave., Greenville, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. James S. Shipman, 542 Cooper St., Camden.
Secretary: Dr. Wright McMillan, 23 Passaic Ave., Passaic.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516-6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

Utah Ophthalmological Society

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd. S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert J. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Harry M. Sage, 9 Buttles Ave., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to June.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St. N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert G. Smith, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St. W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Arthur M. Yudkin, 257 Church St., New Haven, Conn.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. J. T. Maxwell, 1140 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Adolph Krebs, 509 Liberty Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. I. Henry Smith, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter W. Henderson, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. Ernest Sheppard, 927 Farragut Sq. N. W., Washington, D. C.
Secretary-Treasurer: Dr. E. Leonard Goodman, 1801 I St. N. W., Washington, D. C.
Place: Episcopal Eye and Ear Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

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